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The Hand, Part I, Spring, 1959

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Contents

SECTION II

THE PATHOLOGIC PHYSIOLOGY OF METABOLIC BONE DISORDERS

A Symposium
(Conclusion of Section I, Clinical Orthopaedics No. 9)

EDWARD C. REIFENSTEIN, JR., M.D.
Guest Editor

Section III

GENERAL ORTHOPAEDICS

Section II

THE PATHOLOGIC PHYSIOLOGY OF METABOLIC BONE DISORDERS

A Symposium
(Conclusion of Section I, Clinical Orthopaedics No. 9)

Edward C. Reifenstein, Jr., M.D.
Guest Editor

Section III

GENERAL ORTHOPAEDICS

1

Phemister, of Chicago (1882-1951)

EDGAR M. BICK, M.D.*

Dallas Burton Phemister was born on a farm in Carbondale, a rural town in southern Illinois, on July 15, 1882. After a normal boyhood, he went off to college at Valparaiso at the age of 16. He remained there two years, when he moved on to finish his undergraduate work at the University of Chicago, receiving his degree in 1900. Next came Rush Medical College for an M.D. in 1904, followed by an internship at the Cook County Hospital. So ended his schooling. Dr. Phemister then started out quite on his own; immediately he entered the private practice of medicine in LaGrange, near Chicago, and here all resemblance to most young practitioners of his age ends, and the Phemister known to medical literature begins.

Early in his practice he felt drawn to teaching and went to his alma mater as an instructor. In 1908 he became Assistant Clinical Professor of Surgery at Rush Medical College. His ambition led him to the study of pathology, the science then considered to be basic to the practice of surgery. Since it was all but impossible for a serious student to obtain proper instruction in that discipline in the United States, Phemister followed other Americans of his day and in 1909 went off for a two-year period of study abroad. He worked and observed in the hospitals and the laboratories of Paris, Vienna and Berlin.

Upon his return to Chicago he continued his teaching at Rush and entered the surgical service of the Presbyterian Hospital. From 1917 to 1919 he served overseas with his hospital unit as a major in the American Expeditionary Forces of World War I. Returning to Chicago he continued, as before, his practice, investigations and teaching. In 1926 came his first mark of academic distinction. The newly organized Medical School of the University of Chicago was to be opened, and, following the trend of the progressive institutions of the time, a faculty was gathered whose senior officers were to be full-time educators. Phemister, now recognized as one of Chicago's outstanding teachers and surgeons, was offered the Chair of Surgery in 1926. Since the physical facilities of the school would not be ready for students until the following year, Phemister went back to the University College of London for further work.

He remained Professor and Chairman of the Department of Surgery at the University of Chicago for 22 years, and in 1948 became Professor Emeritus. During those years he received many of the academic and surgical honors of American and European societies. Among them were the Presidencies of the American College of Surgeons and of the American Surgical Association. He was elected Honorary Fellow of England's Royal College of Surgeons and member of the Académie de Chirurgie of France. Although in practice he remained a general surgeon, the importance of his work in skeletal surgery and the surgical pathology of bone merited his election as Fellow of the American Academy of Orthopaedic Surgeons.

* New York, N. Y.



Dallas Burton Phemister

Phemister applied himself to studies in several surgical fields, especially during his earlier years. While working at University College in London (1926-1927) he concerned himself with the problems of vascular physiology, especially those aspects related to shock. Somewhat later he became interested, for a while at least, in the pathologic physiology of the gallbladder and the gastro-intestinal tract. Some papers of his earlier period reported passing interest in the surgery of cancer. However, his real contribution by far lay in the surgical pathology of bones. In this field he earned a permanent niche in the annals of American orthopaedic surgery. It is proper that a critique of that work should be published before its references become too deeply embedded in the prodigious skeletal literature of the mid-century.

During Phemister's early years, the century-and-a-half-old problem of bone transplantation or bone grafting was being reexamined actively by Axhausen and Lexer, in Germany, and by MacEwen, in Scotland. Ollier had just completed his extensive studies in France. In 1914 Phemister launched into the subject with his publication on *The Fate of Transplanted Bone.*[1] He was among the first of the American osteologists to do so. Throughout his career he retained an interest in bone grafts, which became the subject of a number of successive reports[13,25] both in the laboratory and operating. Subsequently he combined this interest with his studies in the treatment of bone tumors and described cases of bone resections repaired by grafts.[21,22,24] Of these papers, his early ones were of chief importance, since they served to stimulate interest in the problems of bone transplantation in the United States and acquainted his readers with the advanced work of the time of the men whom he had visited abroad.

Another subject which from the beginning engaged his interest constantly was that of the pathology of bone tumors. Here, however, he followed the lead of Bloodgood, who at the time was inspiring a very active and wide study of these neoplasms. Although Phemister wrote of solitary bone cysts,[7] chondrosarcoma,[11] round cell sarcoma,[12] fibrous osteoma of the jaws[18] and, in general, the treatment of bone tumors,[26] his influence, other than educational, cannot be said to have been seriously contributory, excepting possibly his work on chondrosarcoma. However, in 1920 there appeared the first of a lifelong series of papers on the subject with which his name is inextricably associated—that of avascular necrosis. Axhausen's studies in this field were well known to investigators. The necrosis of bone following infection was a common experience. Axhausen described quiet necrosis in the absence of necrotizing pus and referred to the process as aseptic necrosis, due, as he believed, to the lodgment of miscroscopic emboli, bacterial or other, in the blood channels. These emboli were said to have produced intra-osseous infarcts in the localized

areas served by the affected blood channels and, when in sufficient numbers, caused the effect of general area necrosis. In later years, because of the accepted microvascular nature of the process, the term *avascular* necrosis came to supersede *aseptic* necrosis.

Axhausen further described the potential process of healing by peripheral ingrowth of new vascular channels and new trabeculae, using the term *schleichender Ersatz*. In Phemister's writings this term became the *creeping substitution* of common English parlance. His paper on the comparative studies of dead bone as seen in pathologic specimens and on roentgenograms, published in the *Annals of Surgery* in 1920, was his first important contribution to the subject.[3] It caused considerable comment and review and led to further publications in both the surgical and the roentgenologic literature.[3,9,10,20,27,28] Subsequent recognition of roentgenographic appearances of avascular necrosis in its many phases was due in great measure directly and indirectly to this work

Phemister retained his interest in this problem throughout his life. In his hands the subject was expanded greatly and was applied to the interpretation of lesions hitherto inexplicable or otherwise misunderstood. During the 1920's and the early 30's the group of diseases variously termed Legg-Perthes', Kienböck's, Köhler's, Osgood-Schlatter's and similar lesions were classified as osteochondritis. They were believed to be manifestations of an inflammatory reaction in bone. To Phemister they came to represent localized areas of avascular necrosis. Although this process explained their pathology, it did not explain the etiology. At one point Phemister followed Legg in a belief that streptococci formed the emboli causing the bone changes of Legg-Perthes' disease and in related lesions elsewhere in the skeletons.[10] However, in later reports, the avascular necrosis remained, and the streptococci were lost.[27]

An early and a very important paper was his discussion of radium necrosis in bone published in 1926.[9] In this he suggested the mechanism of radium destruction on a vascular basis. In another paper he noted that avascular changes in articular surfaces could explain the pathogenesis of lesions such as osteochondritis-dissecans.[4] He saw the process as the common denominator of the changes in the head of the femur following fractures of the neck, dislocation of the hip and morbus coxae senilis.[9]

Of particular interest was his report with Kahlstrom and Burton on the bone lesions of caisson disease.[10] A serious explosion near Chicago in 1938 presented an opportunity to study the disease, and Phemister, concentrating on its bone lesions, offered the pathogenic explanation of avascular necrosis caused by emboli of concentrated nitrogen bubbles in the blood stream. Although he and his associates published only one report on the subject, his view has remained fixed in its literature.

It is greatly due to Phemister's recognition of the broad application of the concept of avascular necrosis in bone that in the decades which followed the 1930's this process took its place with the traditional school-taught processes of inflammation, and neoplasia, as a basic phenomenon in bone pathology. It is not enough to say that he derived from Axhausen and Lexer. As far as they wrote, aseptic necrosis was a specific reaction in bone which served to explain certain sharply demarcated lesions. Phemister broadened the applicability of the concept to include a large variety of still-unrecognized affections of bone. His contributions to the literature of roentgenology in this field went far in establishing roentgenographic criteria for its several phases. This led to his becoming co-author of a textbook on diagnostic radiology published in 1941.[23]

In 1933 Phemister published his classic paper, *Operative Arrestment of Longitudinal Growth of Bones in the Treatment of Deformities*.[14] This was the introduction to the now commonly accepted surgical procedure of epiphysiodesis and its derivatives. There was no technical background to this

highly original surgical invention. In his paper he discussed the several attempts which had been made to equalize inequality of length of the lower extremities in deformed children and adults. Following the resections of Rizzoli in 1847, in which equalization was attained by shortening the sound side, Codivilla in 1905 suggested lengthening the shortened limb by traction after osteotomy. During the 1920's several attempts at improving the mechanics of this procedure by Abbott and others increased its fashion. However, neither of these operative technics presented sufficient success to make them acceptable to the standard armamentarium of the orthopaedic surgery of their day. When successful the result was gratifying; too often the risk outweighed the anticipation.

To Phemister, who had spent years studying the nature of growth of long bones[17] and was acquainted with experimental work in epiphyseal growth, it occurred that control of this growth plate might retard the development of the normal limb sufficiently to equalize length without the undue risk of the more daring operations. Furthermore, he had available and quoted recent studies on the rate of longitudinal growth in children. He therefore operated to destroy the epiphyseal growth plate at an age calculated to result in equality of length at the time of skeletal maturity. The concept took root in the orthopaedic literature. Eventually modifications by Blount and others permitted greater leeway in the matter of growth calculations. Stapling superseded complete destruction of the plate and allowed for correction of judgment in the time factor. Whatever the future of these procedures, and later experiments in stimulating activity of the plate on the shorter side may invalidate the older technic, to Phemister is due the concept of equalizing limb length by attacking the physiologic mechanism of epiphyseal growth

Spaced among the papers of his more constant interests were a number of apparently isolated studies of tangential problems in bone pathology. Bone and joint tuberculosis,[15] epiphyseal and articular pressures,[5] and fibrous osteomyelitis (a nonsuppurative form)[8] were among these studies. Several of his papers on tumors, and especially their treatment by resection and bone transplant, had some temporary influence, but his work in the field of bone neoplasms was overshadowed by Bloodgood, Coley, Jaffe and others, to whom bone tumors were a major interest.

Phemister accumulated a bibliography of some 150 papers. Many of these were timely and spanned a rather wide range of subjects. He worked constantly. As in all creative and scientific efforts, from this large schedule certain works have become well ensconced in the literature. Others of perhaps temporary interest in their day have enjoyed less recognition. The following twenty-eight publications are a selective listing of Phemister's papers on skeletal pathology and surgery. In the literature of orthopaedic surgery they merit recollection.

REFERENCES

1. The fate of transplanted bone and regenerative power of its various constituents, Surg., Gynec. & Obst. 19:303, 1914.
2. The recognition of dead bone based on pathological and x-ray studies, Ann. Surg 72:466, 1920.
3. Studies in reduction of bone density, Am. J. Roentgenol. 8:355, 1921.
4. Causes and changes of loose bodies arising from the articular surface of the joint, J. Bone & Joint Surg. 6:278, 1928.
5. The effect of pressure on articular surfaces, Ann. Surg. 80:481, 1924.
6. Radium necrosis of bone, Am. J. Roentgenol. 16:340, 1926.
7. (With J. E. Gordon) The etiology of solitary bone cyst, J.A.M.A. 87:1429, 1926.
8. Chronic fibrous osteomyelitis, Ann. Surg. 90:756, 1929.
9. Repair of bone in presence of aseptic necrosis resulting from fracture, transplantation and vascular obstruction, J. Bone & Joint Surg. 12:769, 1930.
10. (With A. Brunschweig and L. Day) Streptococcal infections of the epiphyses and

short bones and their relation to Legg-Perthes' disease, Kienböck's disease, and Köhler's disease of the tarsal navicular, J.A.M.A. 95:995, 1930.
11. Chondrosarcoma of bone, Surg., Gynec. & Obst. 50:216, 1930.
12. Undifferentiated round cell sarcoma, Ann. Surg. 93:125, 1931.
13. Splint grafts in the treatment of delayed non-union of fractures, Surg., Gynec. & Obst. 52:376, 1931.
14. Operative arrestment of longitudinal growth of bones in the treatment of deformities, J. Bone & Joint Surg. 15:1, 1933.
15. (With C. H. Hatcher) Correlation of pathological and roentgenological findings in the diagnosis of tuberculous arthritis, Am. J. Roentgenol. 29:736, 1933.
16. Fractures of the neck of the femur, dislocation of the hip and obscure vascular disturbances producing aseptic necrosis of the head of the femur, Surg., Gynec. & Obst. 59:415, 1934.
17. Bone growth and repair, Ann. Surg. 102: 261, 1935.
18. Fibrous osteoma of the jaws, Ann. Surg. 105:564, 1937.
19. (With S. C. Kahlstrom and C. C. Burton) Bone and joint changes in caisson disease including impaction and deforming arthritis, Surg., Gynec. & Obst. 68:129, 1939.
20. The pathology of ununited fractures of the neck of the femur with special reference to the head, J. Bone & Joint Surg. 21:681, 1939.
21. Bone transplantation in the treatment of tumors and dystrophies of bone, Soc. internat. de chir. XI cong. (1938), Brussels, 1939.
22. Conservative surgery in the treatment of bone tumors, Surg., Gynec. & Obst. 70: 355, 1940.
23. (With P. C. Hodges and A. Brunschweig) Diagnostic Radiology, New York, Nelson, 1941.
24. Rapid repair of defect of femur by massive bone grafts after resection for tumors, Surg., Gynec. & Obst. 80:120, 1945.
25. Treatment of ununited fractures by onlay bone grafts without screw, or tie fixation and without breaking down of the fibrous union, J. Bone & Joint Surg. 28:946, 1947.
26. Cancer of bone and joint, J.A.M.A. 136: 545, 1948.
27. Cystlike lesion of carpal bones, associated with ununited fractures, aseptic necrosis and traumatic arthritis, J. Bone & Joint Surg. 30A:151, 1948.
28. Treatment of the necrotic head of the femur in adults, J. Bone & Joint Surg. 31A:55, 1949.

Section I

AFFECTIONS OF GROWTH CENTERS (EPIPHYSES AND APOPHYSES)

2

Developmental Deviations in the Carpus and the Tarsus

RONAN O'RAHILLY, M.D.*

The chief developmental deviations found in the skeleton of the hand and the foot include: (1) severe anomalies associated with abnormalities in the forearm or the leg and in the digits; (2) structural alterations associated with deformities such as talipes; and (3) accessory elements, multipartitions, and "fusions" in the carpus, the tarsus or the digits. This chapter deals with the third category and is limited to a consideration of the carpus and the tarsus. The anomalies discussed are of embryologic, radiologic and orthopaedic interest, and possess a medicolegal importance.

TERMINOLOGY

The names of the carpal bones are a perennial trouble to medical students. Although the names of the tarsals date back to classical times (with the exception of the cuneiforms, which are attributed to Fallopius), the carpals were named for the first time by Lyser in 1653.[16] In the 18th century other names were added to the list, and the resulting confusion has persisted down to the present time. Of Lyser's terms, lunatum is the only one now used for the original bone (Lyser's trapezium and trapezoideum having been transposed in error).

There seems to be little further excuse for confusion since the establishment of an internationally accepted anatomic terminology in 1955. The *Nomina anatomica* terms, which will be used throughout this chapter, are listed below. In their preparation, however, it was intended that they be employed in translated form where applicable (Fig. 1).

Ossa carpi: os scaphoideum, os lunatum, os triquetrum, os pisiforme, os trapezium, os trapezoideum, os capitatum, os hamatum.

Ossa tarsi: talus, calcaneus, os naviculare, os cuneiforme mediale, os cuneiforme intermedium, os cuneiforme laterale, os cuboideum.

DEVELOPMENT

The carpus and the tarsus can first be distinguished as condensed mesenchyme at about 5 ovulation weeks of age, in embryos of about 9 to 14 mm. C.R. length. A few days later (12-21 mm.) the individual carpals and tarsals begin to chondrify, with the exception of the pisiform, which appears several days later than the other elements. The carpals and the tarsals chondrify in a definite sequence, but the order is different from that of ossification.[22] Ususally ossification does not begin in the carpus until after birth. The capitate and the hamate, however, may show ossific centers in the newborn.[3] Generally, the individual carpals begin to ossify during the first 6 years, with the exception of the pisiform, which commences ossification at about 10 years.[7] Oc-

* [illegible]

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casionally, some of the carpals, particularly the lunate, the scaphoid and the pisiform, may begin to ossify at two or more loci each.

In the case of the tarsus, ossification commences before birth. An inconstant[33] area of ossification (Fig. 2), described as perichondral[11] or parachondral,[12] may be seen as early as 13 menstrual weeks (95 mm.[23]). It is lateral in position, appearing in a groove between the lateral process of the tuber (lateral tubercle) behind and the peroneal process in front.[12] Thus it is behind and not, as stated frequently, at the site of the peroneal trochlea (trochlear process; peroneal tubercle). Some workers[11] consider that the ossification of the calcaneus is comparable with that of a tubular bone, in that perichondral ossification is followed by the appearance of an endochondral center. Subsequently an endochondral ossific center appears in the calcaneus (118 mm.;[18] 220 mm.[12]). Apparently two such centers may sometimes appear; at any rate, double centers have been illustrated in infants by several writers.[2,29]

At about 5 to 9 years of age a center appears for the tuber of the calcaneus; it consolidates at puberty. In addition, a smaller center for the top of the tuber appears about a year or two before puberty.[8]

The second tarsal which exhibits bone formation is the talus, during the second half of intra-uterine life (235 mm.[20]). Its center, however, is not always present in the newborn.[3] The talus also may present several ossific points.[24]

A center in the region of the lateral tubercle of the posterior process (posterior tubercle) usually appears at 5 to 13 years. In most instances it fuses shortly before puberty. It is said that frequently there is a center for the medial tubercle of the posterior process.[15]

In some cases, the cuboid begins to ossify before birth; in others, postnatally. It is said to develop usually from several foci.[11] The lateral cuneiform rarely presents a center in the newborn,[3] but usually all the tarsals display ossific centers by 2 years.[6] The medial cuneiform and the navicular may

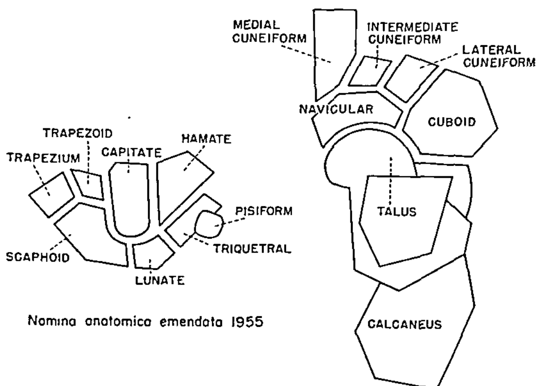


FIG. 1 Scheme of the carpus and the tarsus to show the internationally accepted names of the various elements.

each develop from two centers. An isolated, distal, accessory center for the medial cuneiform has been described as common in children.[14]

Race, sex and weight at birth are factors which have been correlated with the incidence of at least certain ossific centers (e.g., capitate, hamate, talus, cuboid, lateral cuneiform) found in the newborn.[3] The importance of endocrine factors can be gathered from the results of high thyroid dosage in cretinism: in one infant, six carpal ossific centers appeared in the hand at the early age of 15 months.[4]

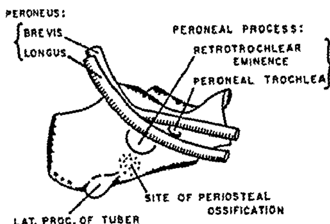


Fig. 2. Lateral surface of the right calcaneus to show the site of periosteal (perichondral or parachondral) ossification and of the peroneal process.

ACCESSORIA

In addition to the eight carpal and seven tarsal bones already listed, and sometimes termed canonical elements, about 60 named inconstant ossicles in the region of the carpus and the tarsus have been recorded. For the sake of brevity and without further implication, these may be called accessory carpal and tarsal elements, or ossa accessoria

Although the inconstant bones since named after him were featured by Vesalius in 1543, scant attention was devoted to the accessoria until the work of Gruber, Thilenius, Pfitzner, Dwight and Grumbach appeared during the last hundred years. Pfitzner's monumental studies were marred, however, by his endeavor to consider each of the accessoria as a bone of evolutionary significance. Traces of this phylogenetic preoccupation are still found in many current reports on anomalies. The accessoria are primarily an ontogenetic problem and

> although they illustrate no phylogenetic principles . . . do make evident the ontogenetic truth that prominent outgrowths of individual bones *often tend to have a separate centre of development* and, as Dwight says, they show "the readiness of the elements of the tarsus to change their shape according to the circumstances."[13]

Comprehensive accounts of the accessoria have been published during the last decade by Marti,[17] Trolle,[33] Werthemann,[34] O'Rahilly[21] and Köhler, as revised by Zimmer.[14] The first two of these works concern only the tarsal accessoria. A very extensive bibliography can be obtained by a combined perusal of these five sources; hence a detailed list of references will not be provided here. The reader should be cautioned that much of the theoretic superstructure added by Trolle is not admissible to the present author. It is highly desirable that writers acquaint themselves with the five works mentioned above before publishing descriptions of new cases. Three of the accounts are available in English.

Definition of Accessoria

The accessory ossicles (of the foot) have been defined[33] as follows:

> Accessory bones are all inconstant, independent, well-defined bones—in an otherwise normally developed foot—the existence of which is not due to a recent minor fracture or other definitely pathological condition, no matter whether these bones bear no, or a less or more intimate relationship to the constant bones, or *entirely replace them because of a division of* the latter into several segments.

Certain of the accessory tarsal ossicles are commonly referred to as sesamoid bones, but generally it is difficult to make a satisfactory distinction between accessoria and sesamoids. In some instances it is diffi-

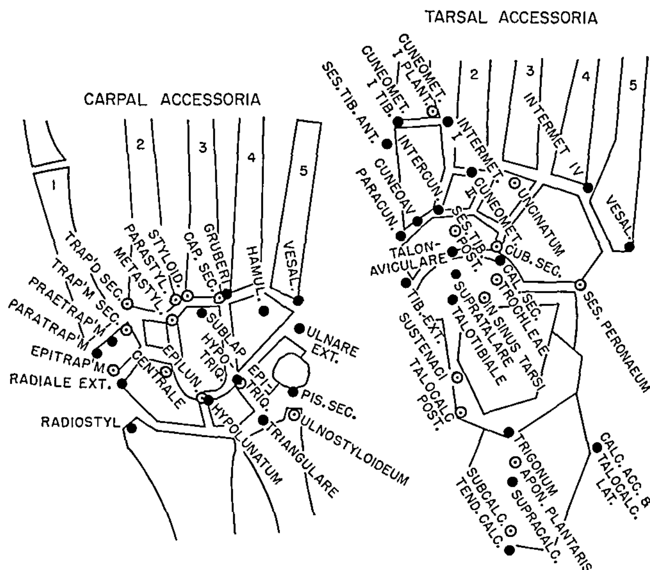


FIG. 3. Scheme of the carpus and the tarsus to show the sites of the various accessoria.

cult also to make a distinction between an accessory element (e.g., tibiale externum) and the smaller constituent of a bipartite bone (e.g , naviculare bipartitum).

Terminology of Accessoria

The nomenclature of the accessoria is even more confusing than that of the canonical elements, and the names employed in standard works (such as that of Kohler[14]) leave much to be desired. Synonyms will be given here, but an attempt will be made to standardize the terminology of the accessoria. The two guiding principles used are: (a) to retain conformity with the *Nomina anatomica;* (b) to avoid, where possible, morphologic terms based on doubtful phylogenetic speculations. As an example of the first principle, epitriquetrum is clearly more informative than epipyramis, talonavicular more in line with modern terminology than astragaloscaphoid. As an instance of the second principle, the term *prehallux* has been scrupulously avoided; the justification for this becomes apparent when it is realized that the tibiale externum, paracuneiforme, cuneometatarsale I tibiale and cuneometatarsale I plantare have each been claimed "to represent" a prehallux. Science should be made of sterner stuff.

The following list of accessoria is arranged alphabetically. Synonyms, many of

which are quite unjustifiable, are placed in parentheses for reference purposes. The letters M., T., W., and K. stand for Marti,[12] Trolle,[33] Werthemann[34] and Köhler,[14] and they refer to the existence of reproductions in these authors' works of radiograms of the relevant accessoria. The locations of the ossicles can be seen from Figure 3.

Ossa Carpalia Accessoria

Os capitatum secundarium (carpometacarpale V). K.
Os centrale (centrale dorsale, episcaphoid). K.
Os epilunatum (centrale II). K.
Os epitrapezium. K.
Os epitrapezoideum (trapezoideum dorsale).
Os epitriquetrum (epipyramis, centrale IV). K.
Os gruberi (carpometacarpale VI). K.
Os hamulare basale (carpometacarpale VII).
Os hamuli proprium. K.
Os hypolunatum (centrale III). K.
Os hypotriquetrum.
Os metastyloideum.
Os parastyloideum (carpometacarpale III).
Os paratrapezium. K.
Os pisiforme secundarium (ulnare antebrachii, metapisoid). K?
Os praetrapezium (carpometacarpale I).
Os radiale externum (parascaphoid). K.
Os radiostyloideum. K.
Os styloideum (carpometacarpale IV). W. K.
Os subcapitatum.
Os trapezium secundarium (multangulum majus secundarium, carpometacarpale II). K.
Os trapezoideum secundarium (multangulum minus secundarium). K.
Os triangulare (intermedium antebrachii, triquetrum secundarium).
Os ulnare externum. K.
Os ulnostyloideum
Os vesalianum manus (vesalii, carpometacarpale VIII). K.

Ossa Tarsalia Accessoria

Calcaneus accessorius (os trochleae). K.
Calcaneus secundarius (calcaneus bifidus, calcaneum surnuméraire, secondary os calcis). M. T. K.
Os aponeurosis plantaris. T.
Os cuboideum secundarium. K?
Os cuneometatarsale I dorsale fibulare.
Os cuneometatarsale I plantare (pars peronaea metatarsalis I, praehallux).
Os cuneometatarsale I tibiale (praehallux). T.
Os cuneometatarsale II dorsale. T. K.
Os cuneonaviculare I dorsale (naviculocuneiforme I dorsale, infranaviculare, paracuneiforme I). T. W. K.
Os infranaviculare. *See* Os cuneonaviculare I dorsale.
Os in sinus tarsi.
Os intercuneiforme.
Os intermetatarsale I (intermetatarseum gruberi). M. T. K.
Os intermetatarsale IV. T. K.
Os naviculocuneiforme I dorsale. *See* Os cuneonaviculare I dorsale.
Os paracuneiforme (praecuneiforme, praehallux, ossicle of Cameron and Carlier). T.
Os peronaeum. *See* Os sesamoideum peronaeum.
Os retinaculi (patella malleoli?). T.
Os sesamoideum peronaeum (peroneale, sesamum peronaeum, cuboideum accessorium). M. T. W. K.
Os sesamoideum tibialis anterior. K.
Os sesamoideum tibialis posterior.
Os subfibulare (talus secundarius, patella malleoli?). M. T. K
Os subtibiale (talus accessorius, talus secundarius, astragalus accessorius, astragalus secundarius, tibiale inferius, os malleoli). T. W. K.
Os supranaviculare. *See* Os talonaviculare dorsale.
Os supratalare (supertalare, talus secundarius, astragalus secundarius, sometimes incorrectly called Pirie's ossicle). T. K.

Os sustentaculi (sustentaculi proprium, subtibiale). T. K.
Os talocalcaneare laterale (talocalcaneus). *See* Calcaneus accessorius.
Os talocalcaneare posterior. T.
Os talonaviculare dorsale (supranaviculare, dorsal astragaloscaphoid ossicle, intertaloscaphoid, taloscaphoid, processus trochlearis of scaphoid or of talus, Pirie's ossicle). M. T. W. K.
Os talotibiale dorsale (talotibiale). T.
Os tendinis calcanei (tendinis achillis). T.
Os tibiale externum (naviculare accessorius, naviculare secundarius, accessory tarsal scaphoid, tibiale anterior, tibiale, praehallux). M. T. K.
Os trigonum (talus accessorius, talus secundarius, astragalus accessorius, astragalus secundaris, intermedium tarsi). M. T. K.
Os trochleae. This term has also been used for the calcaneus accessorius and for the os supratalare.
Os uncinatum (unci, processus uncinatus of lateral cuneiform).
Os vesalianum pedis (vesalii). M. T. K.
Subcalcaneus (os subcalcis, os tuberis calcanei).
Supracalcaneus (os accessorium supracalcaneum). K.

Development of Accessoria

A number of accessoria have been observed in a cartilaginous state in the hands (e.g., by Thilenius) and feet (e.g., by Trolle) of human embryos and fetuses. The present writer has found, for example, the centrale (numerous instances between 17 & 24.5 mm. C.R.), the hypolunate (28 mm.), the triangulare (49 & 75 mm.) and the pisiforme secundarium (50 mm.) as independent cartilaginous elements. In the case of the foot, he has seen a separate intermetatarsale I (45 mm.). Although one writer[33] did not find the trigonum as an independent cartilaginous element, another[30] did so (39 mm.) In a recent observation[10] of a trigonum (80.5 mm.) it is not stated whether or not it was independent.

Because nodules of hyaline cartilage are thus found in the embryo and the fetus at sites corresponding to at least some of those of the postnatal accessory ossicles, it may be concluded that in many instances accessoria have their origin in prenatal life. Not all instances of prenatal accessoria would necessarily have developed further, of course; some might have disappeared. However, in some cases the cartilaginous primordia may develop independent ossific centers and thereby constitute accessory ossicles; in other cases a cartilaginous primordium may fuse with an adjacent canonical element, in which instance an independent ossific center may still develop but will be united to the canonical element by cartilage.

Independent ossific centers at the sites of accessoria have been observed in children. Thus a center at the site of an os tibiale externum has been illustrated[14] in a 9-year-old girl whose mother possessed the ossicle. However, that such centers do not always remain independent can be appreciated from the fate of a bilateral scaphotrapezial ossicle observed in a boy aged 15; the ossicle fused with the scaphoid one year later.[9]

The possibility of post-traumatic ossification must also be considered. Thus, in one patient, a month after contusion of the dorsum of the foot, bone appeared at the approximate site of a dorsal talonavicular ossicle.[17] In another patient, two years after a fracture of the posterior process of the talus from a fall down some steps, nonunion resulted in a smooth-edged appearance which "no longer suggested a fracture, but was characteristically that of the so-called os trigonum"[15] Another possibility is heterotopic ossification in synovial tags.[25] It is well to point out that "there is reason to emphasize the fact that one and the same type of accessory bone need not have the same genesis in different individuals."[33]

Some authors have published histologic data concerning certain accessoria. There is a great need for considerably more work

along this line, particularly when accompanied by the reproduction of good photomicrographs.

GENERAL FEATURES OF ACCESSORIA

The incidence of accessoria is much higher in the tarsus than in the carpus. In one survey of the carpus[21] the incidence was only about 1.6 per cent, whereas another observer recorded only 0.4 per cent. In the tarsus the incidences reported vary from about 25 per cent to 75 per cent. Some cases of accessoria have been shown to be heredofamilial.[19]

It is important to note that the accessoria may be found unilaterally as well as bilaterally. The tarsal accessoria are said to be unilateral in about half the cases, perhaps in even more than half. Several accessoria may sometimes be observed in a single foot, and accessoria are sometimes found to be multipartite. Although dislocation is rare, accessoria may be fractured, either together with fractures of adjacent tarsals or independently; poor repair is usual. Pathologic changes, such as those seen in arthritis, may be observed in accessory elements.

At least some of the accessoria display a peculiar characteristic[5] which we may term *apparent bilocation.* By this is meant that a given ossicle may be found as (a) a free element which, considered together with its adjacent canonical element, appears merely to complete the usual form of the latter, or (b) a free element which appears to be additional to the adjacent portion of its canonical element. An example will make this clear. Thus, the tibiale externum may be found as (a) a free element which appears to be, or to take the place of, the tuberosity of the navicular, or (b) a free element present in addition to a well-developed tuberosity. In other words, the tibiale externum may occur either *as* the tuberosity or merely *at* the tuberosity.

Painful symptoms may arise spontaneously from the presence of tarsal accessoria, sesamoids or "fusions," even in the apparent absence of accidental damage.[17] The tibiale externum is a case in point. On the other hand, tarsal accessoria frequently are mute; i.e., they become manifest and painful only after trauma, which in itself may be very slight. Recovery from an injury may be prolonged because of damage to an accessory element. In individual cases the following question, posed by Marti, assumes a medicolegal significance: "Is the healing process, as the result of an accident, complicated and prolonged through the previous condition of inconstant tarsal elements?"

The precise diagnosis is largely radiologic, and the differential diagnosis may be exceedingly difficult at times. General radiologic criteria should be applied (e.g., the structure and the density of the ossific locus should be considered), and, in doubtful cases, works such as that of Köhler[14] should be consulted. Among the pitfalls may be mentioned the false appearance of an ossicle seen in certain views; e.g., the superimposition of the outline of the lateral cuneiform on that of the navicular may simulate an accessory element distal and lateral to the navicular (*see* Köhler,[14] Fig. 1139). Reliance on only one view may lead to misinterpretation; e.g., in one case a lateral view showed what appeared to be a calcaneus secundarius, but a plantar film demonstrated that the ossicle was actually a tibiale externum (*see* Köhler,[14] Fig. 1092).

MULTIPARTITION

Prescinding from the multipartite scaphoid, concerning which little agreement has been reached, a number of instances of bipartite carpals and tarsals, apparently nontraumatic in origin, have been recorded. The bipartite medial cuneiform is an example.[1] The present writer has seen a bipartite medial cuneiform bilaterally at 18 mm. C.R. It appears, therefore, that bipartition may arise early in intra-uterine life. However, this finding must not be assumed to favor

the idea that the anomaly is "progressive." Many bones have been observed in a bipartite condition, and the number of types of multipartition, accessoria and "fusion" is legion. These multifarious conditions may arise early in prenatal life, as has been shown. They must be accepted as anomalies; they cannot all be assimilated into a phylogenetic theory according to which they are classified as "atavistic" or "progressive."

In cases of apparent multipartition seen in children (e.g., the calcaneus, referred to already) and adults, if only radiographic findings are available, the possibility that several ossific areas are situated in a single cartilaginous element must be considered. As has been mentioned earlier, occasionally some of the carpals and tarsals may begin to ossify in two or more loci each. However, these areas may be merely multiple calcific centers;

anatomically one should speak of two or more independent ossification centers only when resting cartilaginous tissue is found between them. Calcified foci are frequently observed in the roentgenogram where they are often spoken of erroneously as multiple ossification centers. Anatomically, merely a simple ossification center formation is present.[28]

FUSION

By carpal and tarsal "fusion" (Fig. 4) is meant the occurrence as a single structure of that which is usually composed of two (or more) elements. In the case of the foot, talocalcanean and calcaneonavicular fusions, as is now well known, frequently are of clinical importance. In the hand, probably the most common fusion found is the os lunatotriquetrum. This was observed only once in 743 wrists (0.13%) by the present writer, but it has a distinctly higher incidence (6%) in West Africans.[32] Usually there is a distal groove between the lunate and the triquetrum in cases of lunatotriqutral fusion.

Instances of carpal and tarsal fusion have been found in embryos and fetuses. Thus "fusions" between the capitate and the third metacarpal have been seen by the present

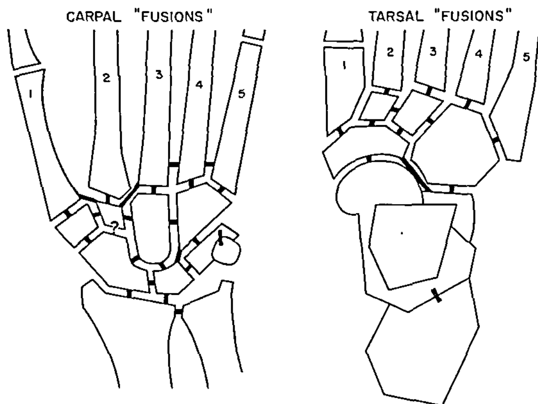


Fig. 4. Scheme of the carpus and the tarsus to show the sites of recorded cases of "fusion"

writer at 24, 28 and 150 mm. C.R., and "fusion" between the lunate and the triquetral at 156 mm. The first two of these cases were observed in hands prior to the occurrence of joint cavitation. It is quite likely, therefore, that many cases of "fusion" arise in the embryo in the cartilaginous carpus or tarsus, and that articular "interzones" and cavities never form at these sites. In other words, such instances arise probably not by fusion but rather by nonseparation of two elements. However, the possibility that interzone formation without subsequent cavitation may take place in some instances must be allowed; this is what usually occurs between the scaphoid and the centrale.

In the case of the foot, talocalcanean and calcaneonavicular unions have been recorded in embryos and fetuses.[10,31]

There is little reason to believe that accessory elements play a role in the development of carpal and tarsal fusions, as has been claimed frequently. However, because the influence of an accessory element may be postulated to take place at cartilaginous stages, postnatal radiographic findings which have been supposed[31] to negative this idea are, in point of fact, completely irrelevant.

After ossification centers have appeared in the individual elements, the existence of a single cartilaginous primordium, where usually there are two, may well favor the subsequent fusion of the two ossific centers concerned. The situation then is somewhat similar to the formation of the scapholunate found in a number of mammals. Thus in the cat[26] and the dog[27] the scapholunate (radiointermediocentrale) develops in what seems to be a single area of cartilage, three bony centers which fuse subsequently.

The progress of fusion of the ossific centers for the lunate and the triquetral has been followed nicely in the hand of a girl by serial radiography.[31]

As in the case of the accessoria, it is not necessary that all instances of fusion have the same mode of development. In addition, the existence of pathologic fusions must be kept in mind.

REFERENCES

1. Barlow, T. E.: Os cuneiforme I bipartitum, Am. J. Phys. Anthropol. 29:95-111, 1942.
2. Caffey, J.: Pediatric X-ray Diagnosis, ed. 3, Chicago, Year Book Pub., 1956.
3. Christie, A.: Prevalence and distribution of ossification centers in the newborn infant, Am. J. Dis. Child. 77:355-361, 1949.
4. Colver, T., and Lodge, T.: Accelerated ossification in a cretin caused by high thyroid dosage, Brit. M. J. 1:89-90, 1952.
5. Dwight, T.: A criticism of Pfitzner's theory of the carpus and tarsus, Anat. Anz. 35: 366-370, 1909.
6. Francis, C. C., and Werle, P. P.: The appearance of centers of ossification from birth to 5 years, Am. J. Phys. Anthropol. 24:273-299, 1939.
7. Greulich, W. W., and Pyle, S. I.: Radiographic Atlas of Skeletal Development of Hand and Wrist, Stanford, Stanford Univ. Press, 1950.
8. Harding, V. V.: Time schedule for the appearance and fusion of a second accessory center of ossification of the calcaneus, Child Develop. 23:181-184, 1952.
9. Hardman, T. G., and Wigoder, S. B.: An unusual development of the carpal scaphoid, Brit. J. Radiol. 1:155-158, 1928.
10. Harris, B. J.: Anomalous structures in the developing human foot, Anat. Rec. 121: 399 (abstract), 1955.
11. Hasselwander, A : Bewegungssystem *in* Peter's Handbuch der Anatomie des Kindes, vol. 2, pp. 403-589, Munich, Bergmann, 1938.
12. Hintzsche, E.: Beitrag zur Entwicklung des menschlichen Fersenbeines, Ztschr. mikr.-anat. Forsch. 21:531-551, 1930.
13. Jones, F. W.: Structure and Function As Seen in the Foot, ed. 2, London, Baillière, Tindall & Cox, 1949.
14. Kohler, A. (rev. by E. A. Zimmer and ed. by J. T. Case): Borderlands of the Normal and Early Pathologic in Skeletal Roentgenology, ed. 10, New York, Grune, 1956.
15. McDougall, A.: The os trigonum, J. Bone & Joint Surg. 37B:257-265, 1955.
16. McMurrich, J. P.: The nomenclature of the carpal bones, Anat. Rec. 8:173-182, 1914.
17. Marti, T.: Die Skelettvarietäten des Fusses, Part 2 of Debrunner and Francil-

lon's Praktische Beiträge zur Orthopädie, Bern, Huber, 1947.
18. Meyer, D. B.: The Prenatal Development of the Skeleton, Thesis, Wayne State Univ., Detroit, 1957.
19. Nissen, K. I.: A study in inherited brachydactyly, Ann. Eugenics 5:281-301, 1933.
20. Noback, C R., and Robertson, G. G.: Sequences of appearance of ossification centers in the human skeleton during the first five prenatal months, Am. J. Anat. 89:1-28, 1951.
21. O'Rahilly, R.: A survey of carpal and tarsal anomalies, J. Bone & Joint Surg. 35A: 626-642, 1953.
22. O'Rahilly, R., Gray, D. J., and Gardner, E.: Chondrification in the hands and feet of staged human embryos, Carnegie Inst. Contrib. Embryol. In press.
23. O'Rahilly, R., and Meyer, D. B.: Roentgenographic investigation of the human skeleton during early fetal life, Am. J. Roentgenol. 76:455-468, 1956.
24. Ruckensteiner, E.: De normale Entwicklung des Knochensystems im Röntgenbild, Leipzig, Thieme, 1931.
25. Saunders, R. L. de C. H.: The os epipyramis or epitriquetrum, Anat. Rec. 84:17-22, 1942.
26. Schaeffer, H.: Die Ossifikationsvorgänge im Gliedmassenskelett der Hauskatze, Morphol. Jahrb. 70:548-600, 1932.
27. ———: Die Ossifikationsvorgänge im Gliedmassenskelett des Hundes, Morphol. Jahrb. 74:472-514, 1934.
28. Schinz, H. R., Baensch, W. E., Friedl, E., and Uehlinger, E. (Eng. trans. ed. by J. T. Case): Roentgen-Diagnostics, vol. 1, part 1, New York, Grune, 1951.
29. Schlüter, K.: Der "Calcaneus bifidus," eine Ossifikationsanomalie des Fersenbeines im Hackenplattfuss, Fortschr. Geb. Rontgenstrahlen 85:720-727, 1956.
30. Scholder, C.: La rotule chez l'embryon humain, Thesis, Neuchâtel, Imprimerie centrale, 1948.
31. Silverman, F. N.: A note on the os lunatotriquetrum, Am. J. Phys Anthropol. 13: 143-146, 1955.
32. Smitham, J. H.: Some observations on certain congenital abnormalities of the hand in African natives, Brit. J. Radiol 21:513-518, 1948.
33. Trolle, D. (trans. by. E. Aagesen): Accessory Bones of the Human Foot, Copenhagen, Munksgaard, 1948.
34. Werthemann, A : Die Entwicklungsstörungen der Extremitäten, vol 9, part 6, of Lubarsch, Henke and Rössle's Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Springer, 1952.

Deviationes Disveloppamental in le Carpo e in le Tarso

Summario in Interlingua

Iste articulo presenta un revista del anormalitates disveloppamental trovate in le skeleto del carpo e del tarso, con referentias particular a lor genese. Le deviationes es principalmente accessorios, multipartition, e "fusion" Le importante fontes recente de information es Marti (1947), Trolle (1948), Werthemann (1952), O'Rahilly (1953), e Kohler in le revision per Zimmer (1956). Attention special es prestate hic al nomenclatura. Le standardisation terminologic del accessorios, de accordo con le *Nomina anatomica* de 1955, es proponite. Le disveloppamento del carpo e del tarso es summarisate, e le embryologia del varie anomalias es presentate. Exemplos de accessorios, de multipartition, e de "fusion" ha essite trovate per le autor in embryones e fetos human de stadios primitive. Iste anormal conditiones deberea esser considerate primarimente como variationes ontogenetic. Assi illos es de interesse a embryologos e etiam o orthopedistas e radiologos.

3

The Oxford Method of Assessing Skeletal Maturity*

ROY M. ACHESON, D.M.†

During the postwar years a longitudinal study of the physical development of over 650 healthy preschool children was conducted in Oxford, England. One of the facets of the developmental process which received special attention was skeletal maturation, and the hand and the knee were chosen as body areas suitable for preliminary study. At the outset Todd's *Atlas of Skeletal Maturation*[26] was used as a standard of reference for the hand; however, experience showed that this work, both in its original version and in the revised edition of Greulich and Pyle,[9] was not wholly suitable for the purpose. The reasons were twofold: first, the pattern of ossification of the English children differed considerably from the standard pattern shown in the Atlases, and, second, the concept of skeletal age, upon which Todd's work is based, seemed to carry with it theoretic difficulties, which will be discussed below. Therefore, a new technic was developed which attempted to provide skeletal maturation with its own scale of measurement. It is the purpose of the present chapter to describe this, the Oxford, method, drawing examples from the hip joint and the pelvis.

SKELETAL MATURATION AS A PROCESS DISTINCT FROM AGING OR GROWING

In the early weeks after conception, cartilage starts to form from the mesenchyme of the fetus. This differentiation of rudimentary cells into definitive tissue is the first stage in the development of the skeleton, the first stage in a complex of processes which will determine whether the child is to be tall or short, broad or slender,[1] ὄρθος παῖς or σκολιὸς παῖς. Skeletal development is best understood if it is realized that three quite separate considerations must be taken into account, namely, that each composite part of the skeleton gets longer, that they all become bone, and finally that time passes. Manifestly, these processes of growing, maturing and aging are integrated because the term *a two year old* brings to mind a child of a certain height who can walk and, per-

* The greater part of the work on the development of the hip joint and the pelvis was performed in the Department of Anatomy, Western Reserve University Medical School, Cleveland, Ohio. I should like to express my deep gratitude to the [illegible]

which they do not agree and, finally, for giving him permission to publish the Brush Foundation data. Dr. Alice Stewart, of the Department of Social Medicine, University of Oxford, very kindly allowed me to assess and publish the Oxford Child Health Survey material, and Dr. Harold Stuart, of the Harvard School of Public Health, gave me free access to his roentgenograms, which I used to check the validity of the indicators and assess the observer error. Mr. E. H. Thornton gave statistical advice, and Dr. Mary Lemon assisted with the preparation of some of the tables.

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haps, talk a little; it may even suggest a roentgenographic picture in which some ossific centers have newly appeared. But they are also dissociable, because there is a very wide range of sizes in perfectly healthy two year olds, and those same ossific centers may not yet be seen, or others may have made a precocious appearance. If the pathologic is included, the achondroplasic, the pituitary giant or the pubertas precox, then it becomes apparent how widely dissociable growth, skeletal maturation and aging can be. Thus, since we are dealing with three distinct variables in the developing human skeleton, we must think of them separately and, more important, *measure* them separately. The variables are represented diagrammatically in Figure 1.

The first of them is increase in size, or growth. Although it is true that remodeling and thickening of bone are carried out by osteoblasts and osteoclasts which are situated under the periosteum, increase in length of the majority of bones is entirely a matter of the proliferation of cartilage cells. *In utero* it probably occurs throughout the length of the cartilaginous skeleton but later is confined to the growth cartilage plates which lie between metaphyses and epiphyses of the long and the short bones, and to the periphery of the round bones. In a series of studies of the rat which have continued over many years, Evans and his group[16,18,21] have published convincing evidence that this process of chondroplasia is influenced profoundly by the growth hormone of the anterior pituitary, and *in vitro* confirmation of their conclusion is to be found in the work of Verdam.[27] Growth is relatively easy to measure in meters or in any other standard of length.

While this increase in size is going on, the cartilage is invaded by calcium and finally is replaced totally by bone. Osteogenesis is not, in the strict sense of the word, a growth process but, on the contrary, is eventually exclusive of growth, for, once the pliable and proliferative cartilage has given way to solid and permanent bone, the faculty for physiologic growth has gone for

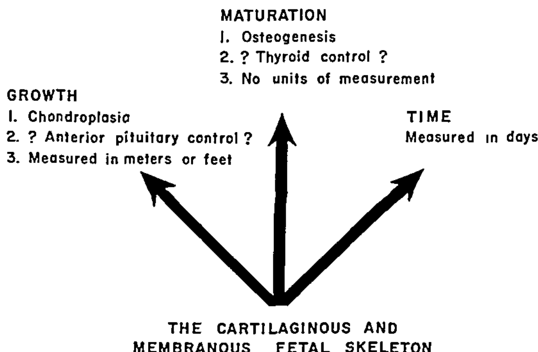


FIG 1. A diagrammatic representation of the three variables to be considered in the assessment of skeletal development, with a note on the possible endocrine control and methods of measurement.

ever. It is therefore appropriate to look upon osteogenesis as a ripening of the skeleton and to describe it as skeletal maturation. In health there is a very close link between maturation and growth, and, although the immediate stimulus to osteoblast activity may come from deteriorating cartilage,[2,11] osteogenesis cannot occur without adequate thyroid function.[16,17] The fact that bone is much more radiopaque than cartilage means that the process of skeletal maturation can be observed in the roentgenogram, and in this way studies can be made of living children.

The third, and final, factor which is essential to the analysis of skeletal development is time, which allows the estimation of *rate* of growth and *rate* of maturation. The measurement of time, like the measurement of growth, presents no fundamental problem. However, this is not true of maturation. Although the process is clear conceptually, its exact measurement in the skeleton or in any other structure or organism is extremely difficult. The commonest method of overcoming the difficulty has been to assume that maturation is so closely correlated with increase in size, or with the passage of time, that it is legitimate to describe the maturity of the structure or organism in terms of its size or its age. For instance, it is still the practice to measure the length of an embryo and from this to deduce its age and maturity. However, Streeter[24] examined the structural differentiation (that is, the maturity) of various organs in a number of embryos and found that the correlation between differentiation and size was not sufficient to justify the assumption He therefore developed his concept of "horizons" and put these forward as a more reliable method of judging the maturity of a small embryo.

In the field of skeletal maturation, Bayley (unpublished data referred to in 1956[5]) has used size as a yardstick for maturity. She has argued that, since the process of skeletal maturation is exclusive of that of growth and both processes are complete at the same time, there must be a constant relationship between the two. She has pointed out, therefore, that knowledge of the maturity of the skeleton at any stage in its development must have some value in the prediction of final height. Thus, although she observes the same indicators on the roentgenogram as Todd and the Cleveland group, she expresses her assessment as percentage of final height attained instead of giving the "skeletal age." However, in a longitudinal study of a group of children followed for the first 20 years of their lives, she has found that the estimated percentage of final height suggested by the roentgenogram and the actual percentage of his final height that the child had achieved at the time were not necessarily the same. Presumably the reason for this was that some dissociation had occurred between the maturation and growth processes.

Time, the third variable, has been used much more commonly as a measure of skeletal maturation, and so the concept of skeletal age has been developed. This approach is no more logical, and, in the last resort, no more accurate than that of using size as the yardstick for maturity. However, although the skeletal age technic is open to this and other criticisms,[1] there is no doubt that the *Atlases of Skeletal Maturity* produced by the Cleveland group[9,10,14,26] provide the best available measure for day-to-day clinical use. Nevertheless, it is equally certain that skeletal maturation must be provided with a yardstick of its own if the processes of physical development are to be fully understood.

THE CLINICAL VALUE OF ASSESSING SKELETAL MATURITY

Thus far it has been shown that skeletal maturation is a distinct entity in the developmental process; however, before going on to describe another technic for its measurement, it may be as well to consider its prac-

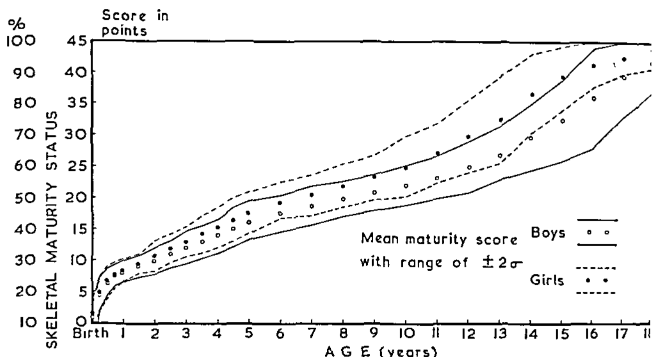


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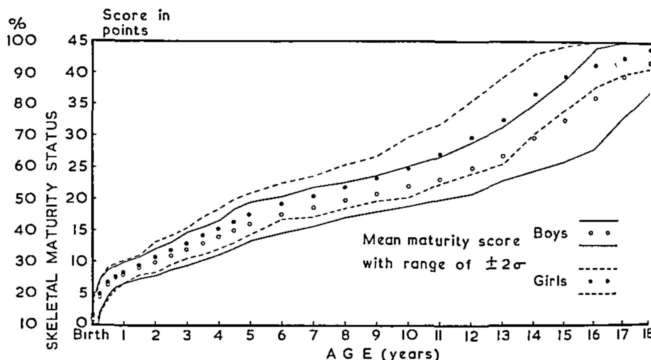


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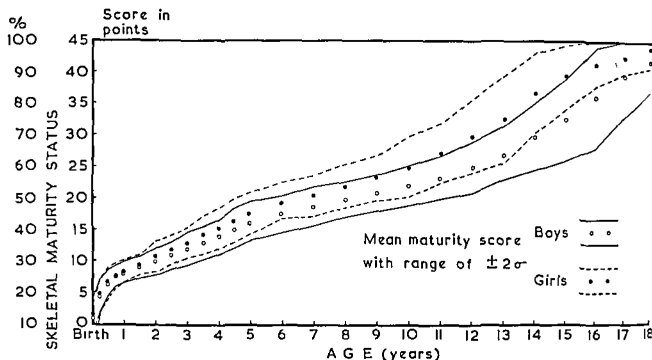


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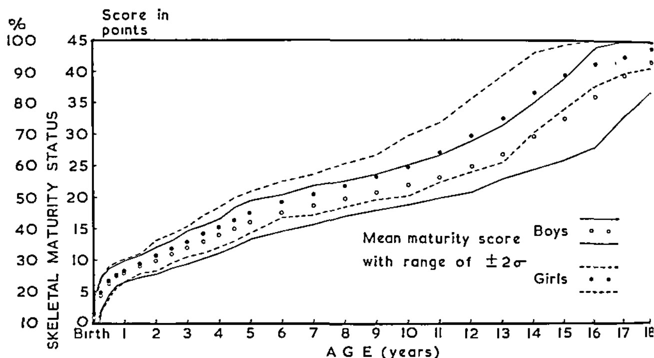


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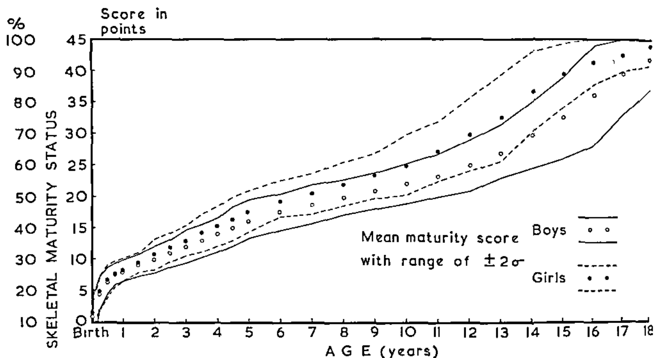


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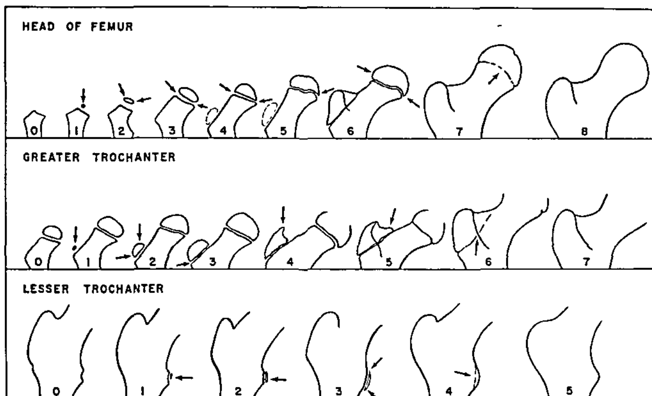


FIG. 3. Maturity indicators at the proximal end of the femur.

Head. (1) Small primitive center, which is usually round in shape. (2) Elongation of the transverse axis. At this stage there often is a tendency to flattening of the aspect facing the growth cartilage plate, with rounding of the acetabular surface. (3) Definition of the length and the depth of the growth cartilage plate. The opposing edges of the epiphysis and the metaphysis now are parallel (4) Reduction of the growth cartilage plate to a thin line with fuzzy, irregular edges. The outline of the articular surface of the epiphysis now is smoothly continuous with that of the metaphysis. (5) The development of a "beak" at the lower medial corner of the epiphysis The growth cartilage plate is no longer a straight line but has acquired a wavy appearance. (6) The epiphysis is wider than the femoral neck and now, on its articular surface, has the contour of the mature bone. (7) The first sign of osseous invasion of the growth cartilage plate. (8) Total ossification of the growth cartilage plate.

Greater Trochanter. (1) Primitive center, which is irregular in outline. (2) Acquisition of a smooth outline. This is rounded in its outer surface and straight in its metaphyseal surface. (3) The outline of the lateral part of the epiphysis becomes aligned and continuous with that of the upper, lateral part of the femoral shaft. (4) An excrescence, or nodule, appears at the upper and medial angle of the epiphysis This nodule may either form as a separate center or develop as an outgrowth of the epiphysis. (5) The filling in with bone of the space between the nodule (indicator 4) and the neck of the femur. (6) The first sign of osseous invasion of the growth cartilage plate. (7) Total ossification of the growth cartilage plate

Lesser Trochanter. (1) Primitive center, which may be linear or rounded. (2) Acquisition of definite linearity. (3) Acquisition of the shape of a "thumbnail cutting," sitting neatly over the metaphysis (4) The first sign of osseous invasion of the growth cartilage plate. (5) Total ossification of the growth cartilage plate. (After Acheson & Dupertuis: Hum. Biol. 29:167)

ated because it has taken on a great deal of the shape of the cartilage. Osteogenesis in the second child is further advanced, and that child's bone is more mature (although it is probably a smaller child).

In the special case of the pelvic girdle, particular care is required in making a distinction between true universal maturity indicators from shape changes which are seen in one sex but not in the other. This is

especially true of the pubic bone, in which a great many such sex-connected characteristics are discernible.[21] Nevertheless, there are many stages in the ossification of all the pelvic bones which do fulfill all the criteria of maturity indicators.

Some maturity indicators are very easy for even the most inexperienced observer to pick out from a roentgenogram, and some are so subtle that they can be seen only by the practiced eye. Some are clearly visible when the bone which is being studied is roentgenographed from one angle and quite indistinguishable when the same bone is roentgenographed in a different plane. However, regardless of the part of the skeleton or of the roentgenographic plane, the indicators for each individual osseous center follow each other in a fixed sequence. This sequence is irreversible because, in health, the process of osteogenesis is irreversible. Once the cartilage has been replaced by bone, there is no going back, and bone it remains.

THE OXFORD METHOD

Study of serial roentgenograms of the skeletons of a number of children, of both sexes and different races, taken through the growing years permits the definition of the particular shape changes that can be described as maturity indicators. For the obvious reason that *change* cannot be recognized in a single roentgenogram of a child, cross-sectional material is quite inadequate for this purpose. If each of the more easily distinguished maturity indicators for any center of ossification is numbered, a maturity scale for that center is constructed. For instance, a series for the head of the femur is shown in Figure 3. Because the process of skeletal maturation is irreversible, a femoral head which is rated at Stage 4 in a roentgenogram has necessarily passed through stages 1 to 3; in fact, it can be described as having scored 4 maturity points. However, it cannot be described as having scored 4 maturity *units*, because this would imply that it was four times as mature as a femoral head in Stage 1, and there is no way of knowing whether or not this is true.* If indicators in other maturing centers in the pelvic region are defined and numbered in this manner, maturity scales can be constructed for them too. And, finally, if all the points for each of these series are added up for any given roentgenogram, the final total score is an index of the skeletal maturity of the region shown in that roentgenogram. This technic, which can be applied equally well to any other developing part of the body, was first described by the author in 1954, when tentative standards were published for the hand and the wrist and for the knee of children up to 5 years of age.[1] Since that time the method has been used successfully for a longitudinal study of over 600 Oxford (England) preschool children[3] and for a similar study of about 300 London children from birth to the age of 3 years.[7] Tanner and his colleagues now are engaged in preparing standards for the hand and the wrist from birth through maturity. In 1956 the present author had the opportunity of setting up standards, hitherto unpublished, of the hip joint and the pelvis,† and these are presented here to serve as a full illustration of the Oxford Method.

HIP AND PELVIS AS A REGION FOR STUDYING SKELETAL MATURATION

There are obvious drawbacks to using the pelvic region in routine assessment of skeletal maturity status. First, it requires a larger roentgenographic plate than any other region of the body and, therefore, is more expensive; second, it entails the exposure of the gonads to radiation (while the risk entailed in the occasional roentgeno-

* In an earlier publication[1] these were described as "Oxford Units," but for the reason stated this was incorrect.

† This research was carried out as Rockefeller Foundation Travelling Fellow in Medicine, and Radcliffe Travelling Fellow of University College, Oxford.

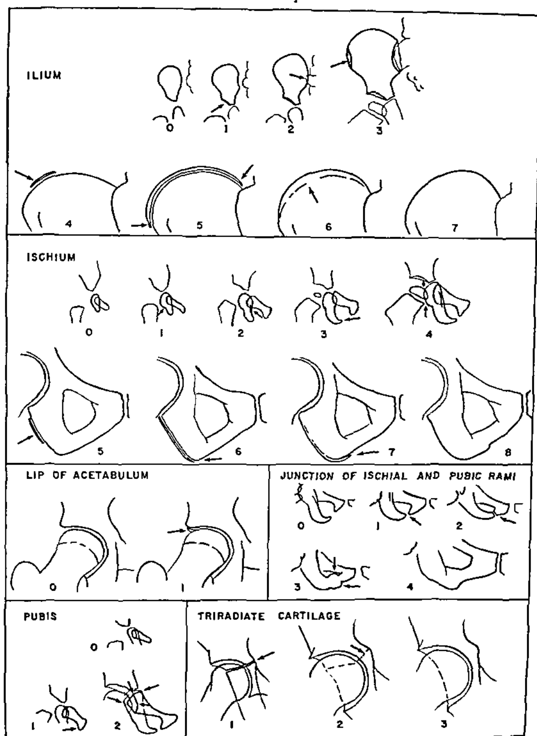


FIG. 4. Maturity indicators in the innominate bone.

Ilium. (1) Early definition of the acetabulum (2) First overlap of ilium and sacrum in the region of the sacro-iliac joint. (3) Definition of the anterior superior iliac spine and the site of attachment of the long head of biceps femoris. According to the position of the individual and to natural variation, this stage may first be detected either as as a region of l ity. Both are indicated in the diagram. pearance of t ary center of the iliac crest. (5) Ossificati crest throug ole length. (6) The first sign of osseous the growth te. (7) Total ossification of the growth carti

Ischium. (1) Appearance of a not ion, on th ct of the ischium (on of the li ensity on r- face of the ischiu uite distinct bular as is and should not b th it. (3) n of the showing as a hoo t the lowe e. (4) nued

TABLE 1. MATURITY SCORES FOR HIP JOINT AND PELVIS FOR OVER 500 AMERICAN AND BRITISH CHILDREN

Age	Boys Total	Boys Maturity Score in Points Mean	Boys S.D.	Girls Total	Girls Maturity Score in Points Mean	Girls S.D.
¼	65	4.3	1.1	40	4.6	1.3
½	134	6.5	1.0	89	6.6	1.2
¾	78	7.5	0.9	58	7.6	0.9
1	138	8.2	0.9	94	8.4	0.9
1½	128	9.2	0.9	108	9.4	0.8
2	133	10.0	1.0	108	10.7	1.2
2½	138	11.0	1.2	112	11.9	1.1
3	141	12.0	1.3	114	12.9	1.3
3½	149	12.9	1.3	115	14.1	1.5
4	161	14.0	1.4	111	15.2	1.6
4½	152	15.0	1.5	116	16.4	1.7
5	157	16.1	1.4	113	17.5	1.6
6	169	17.5	1.5	101	19.1	1.7
7	164	18.7	1.5	101	20.4	1.6
8	164	19.8	1.4	101	21.9	1.7
9	147	20.9	1.4	109	23.4	1.8
10	145	22.0	1.6	126	25.0	2.4
11	116	23.3	1.7	125	27.2	2.4
12	114	25.0	2.1	124	29.9	2.9
13	102	26.8	2.4	96	32.6	3.3
14	86	29.7	2.6	86	36.7	3.2
15	72	32.6	3.3	73	38.3	2.6
16	60	36 1	4.0	65	41.4	1.7
17	41	39.5	3.2	43	42.5	1.4
18	31	41.8	2.1	20	43.6	1.4

FIG. 4 *(Continued from facing page)*
lateral and superior aspect of the ischium and the head of the femur. (5) First appearance of the secondary center of the ischial crest. (6) Ossification of the ischial crest as far as the ischial tuberosity. (7) Ossification of the ischial crest throughout its entire length. (8) Total ossification of the growth cartilage plate.

Lip of Acetabulum. Appearance of the secondary center at the upper part of the lip of the acetabulum. The time span between the first appearance of this center and its fusion is a matter of only a few months.

Junction of Ischial and Pubic Rami. (1) The pointed ends of the two bones are very closely apposed but are not yet in contact. (2) Ossification has proceeded until the outer margins of the bones are smooth and confluent. (3) The development of callus at the site of union. (4) Final union with total absorption of callus and smooth bone margins.

Pubis. (1) Definition of the pubic ramus as a hooklike process at the lower, medial pole of the bone. (2) Ossification of the acetabular portion of the bone. This stage can be recognized both by the definition of the pubic (lower) margin of the triradiate cartilage and of the pubic margin of the acetabulum.

Triradiate Cartilage (the only parts of the cartilage visible in the roentgenographic view which is being studied are those lying between the ilium and the pubis, and the ilium and the ischium). (1) Final definition of the cartilage by total ossification of the three primary centers which surround it. By the time that this occurs the bone margins are ruffled. (2) The first sign of osseous invasion of the triradiate cartilage itself. (3) Total ossification of the triradiate cartilage. (After Acheson & Dupertuis: Hum. Biol. 29:167)

CASE NO. NAME SEX BIRTH DATE

AGE		FEMUR			INNOMINATE BONE						TOTAL		HEIGHT
Yr.	Mo.	Head	Gr. Troc.	Lt. Troc.	Ilium	Isch.	Pubis	*Lip* of Acet.	*Tri-* rad. Cart.	*I-P* Junction	Points	%	

FIG 5 A score sheet suitable for entering assessments of the hip and the pelvis by the Oxford Method

TABLE 2. CORRELATIONS BETWEEN TWO INDEPENDENT ASSESSMENTS OF A SET OF ROENTGENOGRAMS BY THE SAME OBSERVER

Author	Method of Assessment	Part of Body	Age Range of Subjects (Years)	Sex of Subjects	Observer	Number of Roentgenograms	r	z*
Weiner and Thambipillai[28]	Todd[26]	*Hand*	8½ to 20½	Male only	W_1 and W_2	60	0.9942	2.9200
Weiner and Thambipillai[28]	Todd[26]	Hand	8½ to 20½	Male only	T_1 and T_2	60	0.9888	2.5895
Acheson	*Oxford*	*Pelvis*	1 to 18	Both	A_1 and A_2	760	0.9966	3.1875

TABLE 3. CORRELATIONS BETWEEN INDEPENDENT ASSESSMENTS OF A SET OF ROENTGENOGRAMS BY TWO OBSERVERS

Author	Method of Assessment	Part of Body	Age Range of Subjects (Years)	Sex of Subjects	Observer	Number of Roentgenograms	r	z*
Weiner and Thambipillai[28]	Todd[26]	*Hand*	8½ to 20½	Male only	W_1 and T_1	60	0.9899	2.6414
Weiner and Thambipillai[28]	Greulich and Pyle[9]	Hand	8½ to 20½	Male only	W and T	60	0.9772	2.2313
Falkner[8]	*Oxford*[1]	*Hand*	¼ to 3	Both	F and R	806	0.987	2.5143

*$z = \frac{1}{2}[\log_e(1+r) - \log_e(1-r)]$ (Fisher[8])

gram is negligible, it should not be forgotten); and, finally, the child must remove at least some of his clothes. On the other hand, this region has several osseous centers which are suitable for study and, even more important, shows indicators over a very long period. More indicators appear here during the first six months of life than anywhere else in the body, and others continue to appear after the majority of the bones of the body are mature. Thus, practical difficulties are balanced to some extent by definite advantages. It is probably fair to say that roentgenograms of the hip and the pelvis should not be used for maturity assessment when apparently healthy children are being screened, but they should be turned to without hesitation in any case in which diagnosis or interpretation of signs is otherwise difficult, or when a specific assessment of the state of development of the femur is required.

Standards for Hip and Pelvis and How To Use Them

The standards presented in this chapter are the result of about 14,500 assessments of some 8,500 serial roentgenograms of healthy American and British children. The roentgenograms were collected in studies run by the Brush Foundation at Western Reserve University, Cleveland, Ohio, and the Institute of Social Medicine, Oxford, England. The method was also checked against roentgenograms available in the Longitudinal Studies of Child Health and Development at the Harvard School of Public Health.* The position of the child varied slightly in the three series; probably the simplest and most satisfactory is lying supine, with the beam directed just superior to the symphysis pubis The heels should be together, with the legs fully extended and in moderate external rotation. Figures 3 and 4 show the maturity indicators selected for the head of the femur and the pelvis, respectively, and the mean maturity scores with standard deviations are shown by age and sex in Table 1. The way to use the standards is to compare the roentgenograms to be assessed with the indicator series. If there are several roentgenograms of one child taken at increasing ages, it is desirable to display these simultaneously on an illuminated strip. Then the respective indicator series should be studied in turn, noting with the appropriate number each shape change as it occurs. Thus a full point is assigned for each maturity indicator, and the score for the center then remains unchanged until the next indicator is plainly visible. Half points should not be awarded, and no attempt at interpolation should be made. As the indicator series are assessed in turn, it is convenient to enter the scores on a sheet similar to that shown in Figure 5. When all the roentgenograms belonging to a child have been assessed, the total maturity score for each is totaled and placed in the appropriate column on the score sheet. For a truly objective assessment to be made, it is desirable that the assessor should remain in *ignorance of the child's age or height, which* can be entered afterward. Then the scores can be plotted on a chart, such as that shown in Figure 2, thereby enabling the rate of maturation of the child to be compared with the standard.

In order to make the total maturity score comprehensible to a person unfamiliar with the method, it is sometimes desirable to convert it into a percentage * The final adult score of 45 points is inevitable in all healthy people, and so may be accepted as the 100 per cent level. If 0 points is taken quite arbitrarily as 10 per cent mature (that is to say that about 10% of the maturation of the hip joint and the pelvis is completed *in utero*), then all other maturity scores can

* For further details of the sample and methods see Appendix I.

* This device must not be confused with Bayley's system, for, whereas her yardstick is "percentage of predicted final height attained to date," the present scale is a direct measure of skeletal maturity status.

be converted into percentages by multiplying the points value by two.

The pattern variation of the individual indicator series can be studied by using the tables shown in Appendix 2; however, the significance of such variation requires further research before it can be interpreted with confidence.

EVALUATION OF THE METHOD

Observational Error

Two assessors do not always give the same rating of any roentgenogram; nor do two separate ratings made by a single observer necessarily agree with each other. There are several reasons for this. To begin with, although by definition maturity indicators are universal, one indicator may look different in two subjects. For instance, the indicator shown as Number 5 for the head of the femur (Fig. 3) may be very distinct and sharp in one case while in another it may be smaller and rounded. Then these differences may be exaggerated by minor variations in the positioning of the limb, the exposure used and in the manufacture or the developing of the roentgenographic plate. Finally, considerable variation lies with the observer himself, both in his appreciation of the indicators on the roentgenogram and in the scores he assigns to them. The skeletal age technic allows two alternative methods for assigning scores. The first of these, devised by Todd[26] himself, is to make an over-all match between the roentgenogram to be assessed and the standard plate. When the pattern of the two roentgenograms is similar, the assessment is accurate, but when it differs the observer must arbitrarily put more weight on the development of some ossific centers than on others, and so subjective error occurs.

Pyle and her associates[9,15] attempted to overcome this by assigning an exact skeletal age to every bone in the part of the body being examined. In cases where the bones do not match those shown in the standard plates, these assessments are made by interpolation, and this, even to the experienced assessor, is also a subjective and an erroneous procedure.

Mainland[12] compared these two methods and found that, in the hands of an inexperienced observer, Todd's was more accurate than Pyle's. Weiner and Thambipillai[24] also obtained more consistent results using Todd's technic.

The Oxford Method attempts to overcome these errors. No interpolations are ever made, and whole points are awarded only to a center as each distinct shape change occurs, so that the weighting, albeit arbitrary, is intrinsic to the method and, therefore, consistent. It is not left to the observer's own judgment. Nevertheless, it has already been stressed that the shape changes are less distinct in some individuals than in others, and there are times when opinion varies as to the appropriate score.

It is not easy to make a fair comparison between the Skeletal Age and the Oxford Methods because they use different scales of measurement. For instance, Mainland,[12,13] in his excellent analysis of the former technics, expresses the error in terms of "skeletal months," and these bear no constant relationship to Oxford points. In order to overcome this difficulty, the correlation coefficients *r* have been calculated for the data set out by Weiner and Thambipillai[23] and compared with the correlation between two assessments of 760 roentgenograms of the pelvis from the Harvard collection by the present author. These roentgenograms, which are not included in the standards presented in this chapter, were assessed individually and were not laid out simultaneously on an illuminated strip in the manner recommended above. The correlation was higher for the Oxford Method (*see* Table 2) than for Todd's method in the hands of either W. or T., and the difference was significant at the 5 per cent level* for the comparison between A. and T. It is a possibility that

* Fisher's[8] formula $z_a - z_b > 2\sigma_{z_a - z_b}$

TABLE 4. COMPOSITION OF THE SAMPLE

AGE	NUMBER OF BOYS			NUMBER OF GIRLS		
	Cleveland	Oxford	Total	Cleveland	Oxford	Total
¼	65		65	40		40
½	69	65	134	53	36	89
¾	70		70	58		58
1	78	60	138	59	35	94
1½	88	40	128	77	31	108
2	96	37	133	80	28	108
2½	104	34	138	84	28	112
3	108	33	141	86	28	114
3½	111	38	149	90	25	115
4	126	35	161	87	24	111
4½	128	24	152	92	24	116
5	139	18	157	96	17	113
6	169		169	101		101
7	164		164	101		101
8	164		164	101		101
9	147		147	109		109
10	145		145	126		126
11	116		116	125		125
12	114		114	124		124
13	102		102	96		96
14	86		86	86		86
15	72		72	73		73
16	60		60	65		65
17	41		41	43		43
18	31		31	20		20
TOTALS	2,601	384	2,985	2,072	276	2,348

this difference is due to the skill of the observer; almost certainly this is the basis of the superiority of W. over T. Furthermore, there may be technical differences between hand and hip. Therefore, the performance of two assessors using the three methods on the hand is shown in Table 3. Here, although there is no significant difference between the correlations for the Todd and the Oxford Methods, both are significantly higher (at 5% level*) than the Greulich-Pyle Method.

No firm conclusion can be drawn from this analysis as to which of the three technics carries least observer error, but it can at least be said that the Oxford Method compares favorably with the older systems

* See footnote on page 31

In the study of the hip joint and the pelvis, the average difference between two independent assessments was found to be about 0.75 points; less than one reassessment in 40 differed from the original assessment by more than 2 points. The magnitude of the assessment error was independent of sex or of the total score, so that it is reasonable to state that, when the maturity score assigned to one roentgenogram differs from that assigned to another by 2 points or more, there is a significant difference between the *maturity status of those two roentgenograms.*

CONCLUSION

The Oxford Method has a cardinal advantage over any other inspectional method in that it assigns to increasing skeletal maturity

TABLE 5. HEAD OF FEMUR: PERCENTAGE OF CHILDREN (BY SEX) SHOWING EACH OF THE EIGHT DEVELOPMENTAL STAGES AT THE VARIOUS AGE GROUPS (BRUSH DATA ONLY)

AGE	STAGES 1 M	1 F	2 M	2 F	3 M	3 F	4 M	4 F	5 M	5 F	6 M	6 F	7 M	7 F	8 M	8 F
¼	7	38														
½	80	87	10	34												
¾	*99*	*99*	62	79												
1	100	99	84	93		4										
1½		100	99	97	7	23										
2			100	100	20	60										
2½					53	87	1	1								
3					89	96	3	11								
3½					98	100	5	28								
4					100		22	56								
4½							49	85		3						
5							76	98	1	9						
6							90	100	6	28						
7							*100*		*28*	*60*		*1*				
8									54	87		4				
9									86	97	*3*	19				
10									95	99	13	45				
11									98	100	33	68				
12									99		54	89		9		
13									100		78	99		37		10
14											96	100	4	74		39
15											97		34	89		74
16											100		72	*96*	*28*	*86*
17													88	100	50	88
18													*95*		*66*	*95*

a scale of its own. Thus, in contrast with the skeletal age technic, when the maturity score in points is plotted against chronologic age (*see* Fig. 2), periods of rapid change are distinguished from periods of slow change (for this reason the earlier practice[1] of weighting maturity points has been abandoned). Each point is scored when an *obvious* indicator appears on the roentgenogram, so that age periods when the maturity score increases sharply, those when many obvious changes are taking place, are presumably periods of rapid maturation; and, conversely, those years in which there is little increase in score are periods of slow maturation. Furthermore, a comparison of the rate between the sexes is greatly simplified.

The observer error is no higher than that found when skeletal age is used, and there is reason to hope that the Oxford Method will enhance the precision of the estimation of skeletal maturity. However, it is slow to use, in that a certain amount of simple calculation is necessary in each assessment. Thus, in making a quick clinical appraisal of the developmental status of a child in day-to-day pediatric practice, the method of choice is to make an over-all match between the child's roentgenogram and the appropriate plate in the *Atlases* of Greulich, Hoerr and Pyle.[9,10,14] However, in research, or at any time when an exact *measurement* of skeletal maturity of a single bone or a child is required, it is felt that a method, such as the Oxford Method, which allows maturity its own yardstick, has important advantages over the older technics.

TABLE 6. GREATER TROCHANTER (ANALYSIS AS IN TABLE 5)

AGE	Stages 1 M	1 F	2 M	2 F	3 M	3 F	4 M	4 F	5 M	5 F	6 M	6 F	7 M	7 F
¼														
½														
¾														
1														
1½														
2		12												
2½	6	61												
3	27	88		4										
3½	48	97		15		1								
4	66	99	1	49		13								
4½	82	100	8	74		23								
5	97		23	94		60								
6	100		61	99	3	84		5						
7			85	100	7	97		16						
8			99		34	99	2	32						
9			100		70	100	12	69		7				
10					92		31	89		30				
11					100		64	98	8	58				
12							88	99	21	85		19		
13							96	100	50	100		41		12
14							100		90		9	87		34
15									98		30	98	2	72
16									99		70	100	20	91
17									100		88		36	100
18											95		76	

APPENDIX 1

MATERIAL AND METHOD

The exact composition of the sample from which the standards were drawn up is shown in Table 4. Further details as to the purpose and the conduct of the two studies are published elsewhere.[19,20,22]

A single series of Brush Foundation roentgenograms representative of each of the sexes at every age was examined, and shape changes were selected which appeared to fulfill the criteria necessary to be defined as maturity indicators; a total of 54 such changes were found. The roentgenograms of 100 boys and 100 girls, a total of about 3,000 radiographs, then were studied in order to determine which of these changes seemed to be universal, and as a result 45 maturity indicators were chosen (*see* Figs. 3 & 4).

Next, these selected indicators were validated against more than 3,500 roentgenograms in the Harvard collection,[25] and they were all found to be satisfactory. Then the original 200 Brush cases were re-examined, and maturity assessments were made of each; to these were added a further 125 boys and 75 girls, making a total of 400 Cleveland children in all. (These cases do not entirely correspond with the "Research Series" upon whom the *Atlases of Skeletal Maturation* are based.[9,10,14,26])

Some months later the 100 Boston cases with the most complete records were evaluated a second time without any reference to the previous assessments so that observer error could be calculated. Finally, the roentgenograms of just over 100 children in the Oxford Child Health Survey (England) were assessed.

The Cleveland and the Oxford data alone

TABLE 7. LESSER TROCHANTER (ANALYSIS AS IN TABLE 5)

AGE	STAGES 1 M	1 F	2 M	2 F	3 M	3 F	4 M	4 F	5 M	5 F
¼										
½										
¾										
1										
1½										
2										
2½										
3										
3½										
4										
4½										
5										
6		3								
7		18		2						
8	1	35		15		1				
9	6	64	1	34		7				
10	22	84	7	59	1	28				
11	63	98	11	80	8	56		5		
12	78	100	61	97	20	80		16		4
13	97		75	100	63	97		44		13
14	100		98		78	100	14	78		42
15			100		97		53	94	4	67
16					100		79	99	32	90
17							92	100	51	98
18							100		71	100

were available for the preparation of standards. Since the maturity scores in each were very similar and all the children included were considered to be "normal" and "healthy," it was decided that a combination of the two might give the most useful "normal standards."

APPENDIX 2

ANALYSIS OF THE APPEARANCE PATTERN OF THE INDIVIDUAL INDICATOR SERIES

Tables 5 to 11 show the percentage of children in whom each indicator was seen in each of the bones for the first time; this analysis has been made from the Brush data only. The sexes are shown separately, and it is a matter of considerable interest that there is no sex difference in the timing of the appearance of the indicators in the *primary* ossification centers in any of the three composite parts of the innominate bone. *In this* respect this bone differs from any other part of the skeleton of the human or the rat studied by the Oxford Method so far.

REFERENCES

1. Acheson, R. M.: A method of assessing skeletal maturity from radiographs, J. Anat. 88:498, 1954.
2. ———: The response of the developing skeleton *to general systemic* disturbance. In press.
3. Acheson, R. M., Kemp, F. H., and Parfit, J.: Height, weight and skeletal maturity in the first five years of life, Lancet 1:691, 1955.
4. Acheson, R. M., and Macintyre, M. N.: The effects of acute infection or acute *starvation on skeletal* development. In press.
5. Bayley, N.: Individual patterns of development, Child Develop. 27:45, 1956.
6. Falkner, F.: Personal communication.

TABLE 8. ILIUM (ANALYSIS AS IN TABLE 5)

AGE	STAGES 1 M	1 F	2 M	2 F	3 M	3 F	4 M	4 F	5 M	5 F	6 M	6 F	7 M	7 F
¼	100	100	59	57	2									
½			83	81	10	3								
¾			97	97	17	21								
1			100	99	79	87								
1½				100	97	100								
2					100									
2½														
3														
3½														
4														
4½														
5														
6														
7														
8														
9														
10														
11								1						
12								31						
13							13	58		3				
14							42	84	8	23				
15							72	96	29	44		11		
16							94	100	65	73	2	42		7
17							99		70	93	31	56	2	14
18							100		84	100	42	80	16	40

7. ———: Skeletal maturation: an appraisal of concept and method, Am. J. Phys. Anthropol., 1957 (abstract). In press.
8. Fisher, R. A.: Statistical Methods for Research Workers, ed. 12, Edinburgh, Oliver, 1954.
9. Greulich, W. W., and Pyle, S. I.: Radiographic Atlas of Skeletal Development of the Hand and Wrist, Stanford, Cal., Stanford Univ. Press, 1950.
10. Hoerr, N. L., and Pyle, S. I.: Radiographic Atlas of the Skeletal Development of the Foot, Springfield, Ill., Thomas. In press.
11. Lacroix, P.: Organization of Bones, London, Churchill, 1951.
12. Mainland, D.: Evaluation of skeletal age and method of estimating development. Part 1: Systematic error. Pediatrics 12: 114, 1953.
13. ———: *Ibid.* Part 2: Variable error. Pediatrics 13:165, 1954.
14. Pyle, S. I., and Hoerr, N. L.: Radiographic Atlas of Skeletal Development of the Knee, Springfield, Ill., Thomas, 1955.
15. Pyle, S. I., Mann, A., Driezen, S., Kelly, H. J., Macy, I. G., and Spies, T. D.: A substitute for skeletal age (Todd) for clinical use; the Red Graph method, J. Pediat. 32:125, 1948.
16. Ray, R. D., Asling, C. W., Simpson, M. E., and Evans, H. M.: Effects of thyroxin injections on growth and differentiation of the skeleton of hypophysectomized female rats, Anat. Rec. 107:253, 1950.
17. Ray, R. D., Asling, C. W., Simpson, M. E., Walker, D. G., Li, C. H., and Evans, H. M.: Growth and differentiation of the skeleton in thyroidectomized-hypophysectomized rats, treated with thyroxin, growth hormone and the combination, J. Bone & Joint Surg 36A:94, 1954.
18. Ray, R. D., Simpson, M. E., Li, C. H., Asling, C. W., and Evans, H. M.: Effects of the pituitary growth hormone and of thyroxin on growth and differentiation of the skeleton of the rat thyroidectomized at birth, Am. J. Anat. 86:479, 1950.

TABLE 9. ISCHIUM (ANALYSIS AS IN TABLE 5)

AGE	STAGES 1		2		3		4		5		6		7		8	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
¼	97	100	74	86	5	7										
½	100		96	100	39	48										
¾			100		68	68	1	7								
1					91	100	6	14								
1½					100		26	47								
2							63	91								
2½							87	98								
3							95	100								
3½							99									
4							100									
4½																
5																
6																
7																
8																
9																
10																
11										8						
12									1	34		6				
13									9	65		34		3		
14									37	94	9	62		22		
15									67	99	34	94	5	48		
16									81	100	68	99	37	85		
17									95		72	100	53	93	7	18
18									95		89		68	100	17	40

19. Ryle, J. A : Changing Disciplines, London, Oxford, 1948.
20 Simmons, K.: *The Brush Foundation study* of child growth and development. Part 2: Physical growth and development. Monogr. Soc. Res. Child Develop., vol. 9, No. 1, 1944.
21. Simpson, M. E., Asling, C. W., and Evans, H. M.: Some endocrine influences on skeletal growth and differentiation, Yale J. Biol. & Med. 23:1, 1950.
22. Stewart, A. M., and Russell, W. T.: Interim report on the Oxford child health survey, M Officer 88:5, 1952.
23 Stewart, T. D.: Notes on the sexual differentiation of the pubic bone, Am. J. Phys. Anthropol. n.s. 14:388, 1956.
24. Streeter, G L.: *Developmental horizons* in the human embryo, Carnegie Contrib. Embryol. 30:211, 1942.
25. Stuart, H C.: Studies from the Center for Research in Child Development, School of Public Health, Harvard University. I: The Center, the group under observation, sources of information and studies in progress, Monogr. Soc. Res. Child Develop., vol. 4, No. 1, 1939.
26. Todd, T. W.: Atlas of Skeletal Maturation. Part 1: The Hand and Wrist. St. Louis, Mosby, 1937.
27. Verdam, H. D.: Skeletverbeening en differentiate van spiervezels in gekweekte embryonale rattenextremiteiten en de invloed hierop van "groeihormoon" uit de hypophysevoorkwab, Alphen a d. Rijn, Haasbek, 1946. (Cited by Lacroix, *loc. cit.*)
28. Weiner, J. S., and Thambipillai, V.: Skeletal maturation of West African Negroes, Am. J. Phys. Anthropol. n.s. 10:407, 1952.

BIBLIOGRAPHY OF THE OXFORD METHOD (OTHER THAN THAT REFERRED TO IN THE TEXT)

Acheson, R. M.: A method of assessing skeletal maturity without resorting to the concept of skeletal age, Anat. Rec. 124:250, 1956.
Acheson, R. M., and Dupertuis, C. W.: The

relationship between physique and skeletal maturation in boys, Human Biol. 29:167, 1957.

Falkner, F.: Some effects of illness on child development. In press.

Hewitt, D.: Some familial correlations in height, weight and skeletal maturity, Ann. Human Genet., vol. 22, 1957. In press.

Hewitt, D., Westropp, C. K., and Acheson, R. M.: Effect of childish ailments on skeletal development, Brit. J. Prev. Soc. Med. 9:179, 1955.

Macintyre, M. N., Acheson, R. M., and Oldham, E. C.: Techniques in longitudinal studies of the skeletal development of the rat, Anat Rec., 1957. In press.

Le Methodo Oxford de Evalutar le Maturitate Skeletal

Summario in Interlingua

Al minus tres factores variabile es a considerar in le evaluation del disveloppamento skeletal. Le prime es augmento in longor, i.e., crescentia, que in le majoritate de ossos depende del proliferation de cellulas cartilaginose in le lamine epiphysee. Durante iste processo, osteogenese occurre in le epiphyses e in le metaphyses del ossos longe, de maniera que ultimemente tote le cartilagine es reimplaciate e le skeleto es completemente matur e adulte. Iste metamorphose de cartilagine in osso—le secunde factor—es appellate maturation skeletal. Crescentia e maturation es assi processos distincte—ben integrate sub conditiones de sanitate sed dissociabile sub conditiones pathologic. Le tertie factor es le passage del tempore con que le individuo deveni plus vetere. Le inveteramento pote etiam esser dissociabile ab crescentia e maturation. Le passage de tempore e le augmento in longor es relativemente facile a mesurar, sed le maturation ha nulle immediate standard de mesuration

Le maturation skeletal es un processo irreversibile. Le indicios de maturitate in omne osso es visibile in le radiographo, e illos se succede in un sequentia fixe. Assi, quando le plus obvie indicios de un osso es numerate, le presentia del indicio 4 inevitabilemente significa que le indicios 1 a 3 ha jam apparite. Si tal indicios es clarmente definite e numerate, lor *summa total pro* un gruppo particular de ossos es un mesura del maturitate de ille parte del skeleto. In plus, iste valores de alicun indicationes del maturation que varia in relation al avantiante etate del infante. Un comparation directe pote esser facite inter le stato de maturitate skeletal de pueros e de pueras, e le "limites del norma" pote esser estimate statisticamente.

Le standards de iste methodo—le methodo Oxford—pro evalutar le maturitate skeletal es presentate pro le coxa e le pelve. Le standards es basate super 8,000 radiographos de circa 600 pueros e pueras american e anglese qui ha essite studiate serialmente ab le infantia usque al maturitate.

TABLE 10. PUBIS (ANALYSIS AS IN TABLE 5)

AGE	STAGES 1 M	1 F	2 M	2 F
¼	91	100		
½	98			
¾	100			
1				
1½				
2			3	4
2½			8	9
3			14	19
3½			32	43
4			56	59
4½			81	86
5			95	97
6			97	100
7			98	
8			100	
9				
10				
11				
12				
13				
14				
15				
16				
17				
18				

TABLE 11. (ANALYSIS AS IN TABLE 5)

Age	Lip of Acetabulum M	Lip of Acetabulum F	Junction of Ischial and Pubic Rami Stages 1 M	1 F	2 M	2 F	3 M	3 F	4 M	4 F	Triradiate Cartilage Stages 1 M	1 F	2 M	2 F	3 M	3 F
¼																
½																
¾				1												
1			1	4												
1½			9	11												
2			21	23	1	2										
2½			35	36	6	4	1									
3			49	57	11	8	3	1								
3½			67	69	21	19	4	5	1							
4			78	74	31	28	7	11	2	1	1	3				
4½			89	82	51	40	15	17	2	4	6	12				
5			93	88	67	54	24	27	3	5	12	28				
6			95	90	83	71	50	47	11	11	21	40				
7			97	93	93	82	69	62	20	17	47	58				
8			98	96	95	86	84	76	34	30	69	76		1		
9			100	97	98	88	92	84	52	46	79	91		5		
10	4	7		100	99	95	95	88	65	64	92	96		20		
11	6	32			100	98	98	91	80	74	97	97	2	52		1
12	13	73				100	100	96	89	86	98	100	20	84		21
13	30	93						99	90	94	100		46	98		52
14	70	100						100	95	98			81	100	12	90
15	89								99	100			96		42	98
16	98								100				100		82	100
17	99														93	
18	100														98	

Nos conclude que le Atlantes de Maturation Skeletal de Greulich, Hoerr, e Pyle debe esser empleate in le practica pediatric quotidian pro facer un rapide evalutation del radiographo. Tamen, in le recerca o quandocunque un exacte mesuration del maturitate skeletal de un osso o de un infante es requirite, un methodo como le methodo Oxford, que offere pro le maturation skeletal su proprie standards de mesuration, ha distincte avantages super le technicas previemente disponibile.

4

Observations on the Growth of the Female Adolescent Spine and Its Relation to Scoliosis*

I. J. CALVO, M.D.†

As poorly understood as it has proved resistant to treatment, idiopathic scoliosis has fascinated and baffled medical men through the centuries. Efforts to deal with the problem have always been handicapped by a lack of knowledge of what produces it, and the development of a completely successful method of treatment must await understanding of the etiology.

For over 100 years the association between scoliosis and vertebral growth has been described by various authorities. Some have maintained that the development of deformity was limited to the growth period in general, while others have stressed its relationship more particularly to the rapid growth period.[6] Therefore, the importance of predicting both duration and rate of vertebral growth is evident. Risser and Ferguson[8] found that, on an average, vertebral growth ceased at 14 years and 3 months in females. Later, Risser[7] stressed the need for physiologic rather than chronologic points of reference in predicting growth cessation and stated:

> The completion of the ossification excursion of the iliac apophyses has been proven to occur simultaneously with the completion of vertebral growth and with it the curve is static.

This appears to be the clinical consensus; however, the author is unaware of any critical study to support this opinion.

An attempt will be made in this chapter to answer the following questions: What part does growth play in idiopathic scoliosis? Do curves cease to increase before the termination of "the rapid growth" of adolescence? What guides can be used to indicate the growth activity of the spine? Of what value can they be?

MATERIAL

Seventy-two adolescent females were found who were suitable for study after reviewing the material from the growth laboratory at the Newington Home and Hospital for Crippled Children. All had periodic roentgenograms of the spine and the hands, among other studies, through an average follow-up of 3 years and 11 months:

Females with normal spines	27
Females with idiopathic scoliosis, not fused	17
Females with idiopathic scoliosis, fused	28
Total	72

The average time span between successive roentgenograms of individual patients was 10 months, although ranging from 6 to 14 months.

PROCEDURE

Chronologic age, skeletal age[1] and standing height of the patients at the time of every

* Research material from the Growth Laboratory, Newington Home and Hospital for Crippled Children, Newington, Conn, sponsored by C W. Goff. M D, Visiting Orthopaedic Surgeon.

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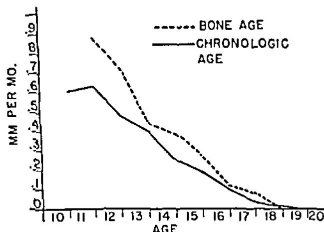


FIG. 1. Growth rate D8-D12 segment. This curve contains about 30 determinations per age group. The short lines between the numerals at the axis of the abscissa indicate that the ordinates represent the time span between the inscribed ages. For example, the ordinate elevated between age 12 and age 13 represents the twelfth year of life or of skeletal maturation, depending on which curve is studied.

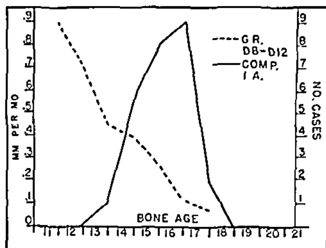


FIG. 2. The curve of incidence of completion of the ossification process at the iliac apophyses is represented for comparison with that of the growth rate. Both curves are based on skeletal age. The growth rate curve does not have the same shape as that of Figure 3 due to the fact that a different scale was used for the ordinates.

spine roentgenogram, as well as the limits and the values of the major curve,[2] were recorded On each roentgenogram the height of a spinal segment, selected as characteristic of the entire vertebral column, was measured in a particular manner. This consisted of the eighth, ninth, tenth, eleventh and twelfth dorsal vertebrae, D8-D12 segment.

On the roentgenograms of normal spines, each one was marked by drawing a line over the inferior margin of the body of the twelfth dorsal vertebra and another over the superior margin of the eighth dorsal. The distance in millimeters between these two lines was recorded as the height of the D8-D12 segment.

On the scoliotic spines, the upper and the lower limits of the segment were determined in the same manner as described above. Then, with a small caliper, the distance between these two lines was measured along the concave and also the convex margins of the vertebral bodies, as well as along a line equidistant to these two margins. These three determinations were averaged, and the result was taken as the height of the D8-D12 segment.

In addition, the following data were compiled:

1. A growth rate or differential height per month at the D8-D12 segment, calculated for every interval between successive roentgenograms, as well as the chronologic and the skeletal age at which this growth rate became equal to zero

2. The chronologic and the skeletal age at the approximate time of inception and completion of the ossification of the iliac apophyses, as well as the time span between them

3. The chronologic and the skeletal age at menarche

All data recorded were divided for analysis into eleven chronologic age groups, ranging from 10 to 21 years, and into eight skeletal age groups, ranging from 10 to 18 years.

RESULTS

The mean growth rates calculated according to chronologic and skeletal age form two roughly parallel curves. They descend gradually from values of 0.60 and 0.90 mm. per month, respectively, at the younger age

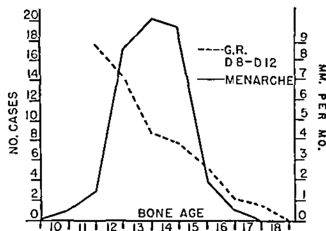


FIG. 3. The curve formed by the age group incidence of the onset of menarche is represented for comparison with that of the growth rates. This curve has a wide peak and a base still wider, which readily suggests the poor value of the onset of menarche as a growth indicator.

groups to zero when skeletal maturity is reached (Fig. 1).

The highest incidence of completion of ossification of the iliac apophyses occurred at a skeletal age of 16 years, when the mean growth rate was around 0.10 mm. per month (Fig. 2).

The highest incidence of onset of menarche occurred at a skeletal age of 13 years, when the mean growth rate was about 0.40 mm. per month. However, the peak of the curve was wide and comprised values ranging from 0.70 to 0.30 mm. per month (Fig. 3).

Using the onset of menarche of our subjects as a point of comparison, it was found that the inception of the ossification process of the iliac apophyses occurred in 44 per cent of the cases in the second year after the onset of the menses. There were also two sizable and almost equal groups, 28 and 25 per cent, respectively, in which it began in the year after or the year before (Fig. 4).

The completion of the ossification process was also correlated in the same way. It was found that it occurred with the same frequency in each of the three years after the onset of the menses (Fig. 5).

Upon analyzing the interval between the inception and completion of the ossification of the iliac apophyses, it was found that 83 per cent of the cases required roughly a year to a year and a half to complete the process (Fig. 6).

DISCUSSION

It was found that the stature determinations in the series were less reliable than

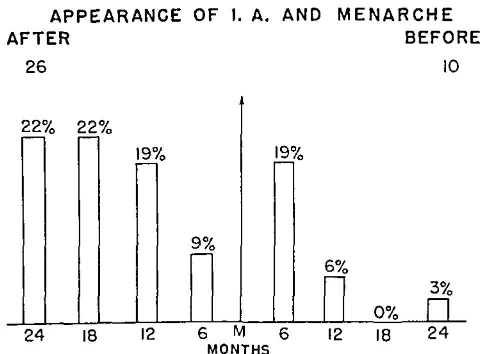


FIG. 4. The inception of the ossification process at the iliac apophyses in 36 patients of the series is compared with the onset of menarche. In most cases, 72 per cent, the ossification begins after the onset of menarche, and in the biggest group, 44 per cent, starts in the second year after the onset of menarche.

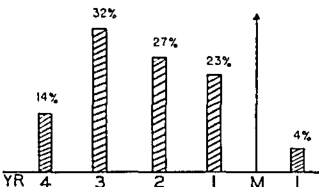


FIG. 5. The completion of the ossification process at the iliac apophyses is compared with the onset of menarche. There is a little difference between the number of cases that finish the ossification process in the first, the second and the third years after the onset of menarche.

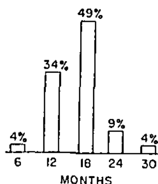


FIG. 6. The time interval between the inception and completion of the ossification process at the iliac apophyses seems to be around a year or a year and a half. The illustration covers 32 cases. It should be pointed out that the later the ossification starts the shorter it takes fully to ossify, and vice versa. This explains the apparent lack of relation between Figures 6 and 7.

what had been hoped for, as various technicians had recorded the height at the time that roentgenograms had been taken. For greater accuracy this otherwise valuable determination was deleted, although the size of the series and the number of measurements would probably have canceled out any error.

Routine anteroposterior roentgenograms of the spine from the growth laboratory were found to be acceptable for study. They had been taken with a standardized technic and were regarded as accurate.

It would have been preferable to measure the whole spine, but, in order to minimize distortional errors, the segment formed by the five lower dorsal vertebrae, D8-D12 segment, was selected. This particular section of the spine forms a very flat curve in the sagittal plane and is nearest to the cassette when the roentgenograms are taken. It is also situated in the central part of the roentgenogram, being in the optimum position regarding the central ray An added value to this choice is the fact that it is involved in most cases of idiopathic scoliosis.[5]

The findings at the D8-D12 segment were presumed to give an adequate representation of the growth activity of the entire spine and were translated into a growth rate (millimeters per month) in order to make them more meaningful in terms of growth activity. It was found that when discrepancies existed between the chronologic and the skeletal age of an individual, the growth rate was consistently in accordance with the latter, as may be seen in the following examples:

CASE 1. In June, 1952, this girl had a chronologic age of 13 years and 10 months, a bone age of 16 years and a D8-D12 segment of 142 mm. If one were to follow the indications of her chronologic age, one would expect her to continue to grow, but 25 months later—July, 1954—the D8-D12 segment was still 142 mm. (Fig. 7, *left*)

CASE 2. In July, 1949, this girl had a chronologic age of 15 years and 3 months, a bone age of 13 years and 6 months and a D8-D12 segment of 126 mm. One would not expect much growth in a girl of this chronologic age, but, in August, 1950, her D8-D12 segment measured 133 mm., or a growth rate of 0.53 mm. per month. Nine months later—in May, 1951—she had grown another 3 mm. or a growth rate of 0.33 mm. per month. These

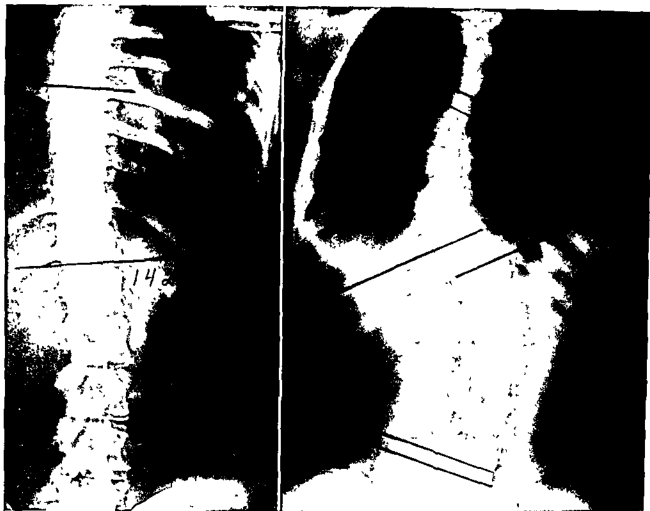


FIG. 7. *(Left)* Case 1. Roentgenograms taken 25 months apart and superimposed to demonstrate absence of growth at the D8-D12 segment. On the earlier roentgenogram the patient was 13 years and 10 months old, but her skeletal age was 16 years. The lumbar vertebrae do not superimpose due to the mobility of the lumbar spine and distortion. *(Right)* Case 2. Roentgenograms taken 22 months apart and superimposed demonstrate marked growth at the D8-D12 segment On the earlier roentgenogram the patient was 15 years and 3 months old, but her skeletal age was only 13 years and 6 months. Note that although the iliac apophyses are complete (the one on the right corresponds to the earlier roentgenogram and that visualized on the left to the later), the patient grew 10 mm at the D8-D12 segment There are a right dorsal (D6-D11) curve of 50° and a left lumbar (D11-L4) curve of 56°, which have maintained their limits and values in spite of marked growth.

findings were consistent throughout with her skeletal age and not with her chronologic age. (Fig. 7, *right*)

For this reason one should always determine the skeletal age of a scoliotic patient. When it corresponds with the chronologic age it will not furnish any added information, but when it does not correspond, growth activity will follow the skeletal age more closely than the chronologic.

A point to be emphasized is that the growth rate curves in Figure 1 are formed by the mean computed for each particular age group. In the individual case its particular curve may be above or below that of the mean, but would be roughly parallel to it. From this standpoint these curves have a clinical application, as they permit one to predict within reason the remaining growth activity of any adolescent female from one single determination.

Considering now the relationship of the

progress of the iliac apophyses to the growth of the spine (Fig. 2), the peak of the curve of incidence of completion of the ossification of the iliac apophyses occurred at a bone age of 16 years with average growth rates of about 0.10 mm. per month. However, in some cases ossification was completed at an earlier bone age, when growth rates of about 0.30 mm. per month were the average. From the individual analysis of these discrepant cases it was concluded that growth activity followed more closely the indication of the bone age than that of the iliac apophyses when a difference existed.

Upon studying the incidence of menarche (Fig. 3), it did not seem that sexual maturity in the female was very closely related to skeletal maturation. In this series menarche occurred at the average age of 12 years and 9 months, which is in accordance with most published figures, but the individual variations were so great as to make this particular criterion valueless.

The interval between the inception and completion of ossification of the iliac apophyses was found to be an additional indicator by which one could evaluate a patient further, but it did not seem to have any advantage over the determination of the skeletal age, which is considerably more accurate and easier to determine.

The different indicators and their value regarding growth prediction at the spine have been discussed. The really important matter, however, is the information one can derive from them when studying scoliotic patients. It is evident that some scoliotic patients, in spite of continued growth of their spines, do not progress to greater deformity, as seen in Figure 7, *right,* so that one may say that growth alone is not the primary factor in the progression of idiopathic scoliosis. It seem to be only the instrument by which other causes, unknown at this time, produce deformity and make themselves evident.

It is postulated here that one of the unknown factors may be a localized or segmental epiphysitis with a different growth rate on one side as compared with the opposite. Once the epiphysitis subsides, the spine resumes its normal growth. Whether or not this is due initially to pressure, as pointed out by Arkin[1] and Nachlas,[4] still remains to be determined.

It was found in the cases of this series that the progression of scoliosis was always associated with growth rates of at least 0.30 mm. per month. However, growth rates higher than this were not necessarily associated with progressive curves.

So far as the value of the completion of the iliac apophyses as an indicator of cessation of progression of scoliotic curves is concerned, it is clearly valid, but in this series more than half the patients had nonprogressive curves one year or more before this time, which makes one feel that it is a somewhat late sign. On the other hand, all these patients had nonprogressive curves at the skeletal age of 15, and only one out of four (23.1%) showed evidence of progression during the skeletal ages of 13 and 14 years. The skeletal age of 15 is reached when the epiphyses of all the long bones of the hand appear to be fused to their respective shafts. The value of this finding is that it permits one to predict the static character of a scoliotic curve as soon as a maturation plate of the hands shows a skeletal age of 15 years. In 35 per cent of the patients this occurs about a year earlier than the criterion described by Risser.

SUMMARY

An attempt has been made to determine the value of various maturation guides such as skeletal age, growth rate of the spine, iliac apophyses and menarche in relation to the prognosis of scoliosis. For this purpose, normal individuals as well as those with scoliosis were studied to determine mean growth rate values, thus establishing a baseline for comparison. A segment of the spine from the eighth to the twelfth dorsal vertebra, D8-D12 segment, was selected, and the growth rate of this segment was determined in all cases. From these values curves were

plotted, aiding in the analysis of the different indicators.

CONCLUSIONS

1. The growth rate of the female adolescent spine is easily determined and is a useful index in evaluating the remaining growth of the spine.

2. In the study of spinal growth, chronologic age seems to be an inadequate measure, and skeletal age should be used.

3. Growth does not cease in the spine in all instances in which the iliac apophyses complete their ossification.

4. For practical purposes, growth activity ceases in the spine at a skeletal age of 16 years; when growth rates are in the vicinity of 0.10 mm. per month.

5. Menarche is not an adequate indicator of spinal growth activity.

6. Scoliotic curves do not necessarily continue to increase while there is growth activity in the spine.

7. Scoliotic curves do not increase when the growth rate of the D8-D12 segment is under 0.30 mm. per month.

8. Progression of many scoliotic curves ceases before completion of the iliac apophyses.

9. Scoliotic curves do not increase after the skeletal age of 15 years, and in 35 per cent of the cases this skeletal age appears a year earlier than the completion of the iliac apophyses.

REFERENCES

1. Arkin, A. M.: The mechanism of structural changes in scoliosis, J. Bone & Joint Surg. 31-A:519-527, 1949.
2. Cobb, J. R.: Outline for the study of scoliosis *in* Am. Acad. Orthop. Surgeons Instructional Course Lectures, vol. 5, pp. 261-275, 1948.
3. Greulich, W. W., and Pyle, S. I.: Radiographic Atlas of Skeletal Development of the Hand and Wrist, California, Stanford Univ. Press, 1950.
4. Nachlas, I. W.: The cure of experimental scoliosis by directed growth control, J Bone & Joint Surg. 33-A:24-32, 1951.
5. Ponseti, I. V., and Friedman, B.: Prognosis in idiopathic scoliosis, J. Bone & Joint Surg 32-A:381-395, 1950.
6. Risser, J. C.: Acquired scoliosis *in* Nelson's Loose Leaf Surgery, vol. 3, pp. 439-451, 1941.
7. ———: Important practical facts in the treatment of scoliosis *in* Am. Acad. Orthop Surgeons Instructional Course Lectures, vol. 5, pp. 248-260, 1948.
8. Risser, J. C., and Ferguson, A. B: Scoliosis: its prognosis, J. Bone & Joint Surg. 18: 667-670, 1936.

Observationes in Re le Crescentia del Columna Vertebral del Adolescente Feminin e in Re su Relation a Scoliosis

Summario in Interlingua

Un studio esseva interprendite pro determinar le valor de varie indicios de maturation—le etate skeletal, le crescentia del columna vertebral, le completion del ossification del apophyses iliac, le menarche—in relation al prognose de scoliosis.

Esseva studiate individuos normal e etiam individuos con scoliosis. Le valores del crescentia medie esseva determinate pro obtener un base de comparation.

Un segmenta del columna vertebral ab le octave al dece-secundo vertebra, D8 a D12, esseva seligite e le rapiditate de crescentia de iste section esseva determinate in omne casos Ab iste valores esseva delineate curvas que esseva utile in le analyse del varie supra-mentionate indices.

Le conclusiones de iste studio es le sequentes:

1. Le rapiditate del crescentia del columna vertebral del adolescente feminin es facilemente determinabile e es un indice utile in evalutar le remanente crescentia del spina.

2. In le studio del crescentia del columna

vertebral le etate chronologic pare esser un mesura inadequate; le etate skeletal debe esser empleate.

3. Le crescentia del columna vertebral non cessa in omne casos con le completion del ossification del apophyses iliac.

4. Le activitate de crescentia in le columna vertebral practicamente cessa al etate skeletal de 16 annos, quando le rapiditate del crescentia es approximativemente 0,10 mm per mense.

5. Le menarche non es un indice adequate del activitate del crescentia del columna vertebral.

6. Le curvas scoliotic non necessarimente continua augmentar se dum le activitate de crescentia continua in le spina.

7. Le curvas scoliotic non se augmenta quando le rapiditate del crescentia del segmento D8 a D12 es infra 0,30 mm per mense.

8. Le progression de multe curvas scoliotic cessa ante le completion del apophyses iliac.

9. Le curvas scoliotic non se augmenta post le etate skeletal de 15 annos; in 35 pro cento del casos isto etate skeletal ocurreva un anno ante le completion del apophyses iliac.

plotted, aiding in the analysis of the different indicators.

CONCLUSIONS

1. The growth rate of the female adolescent spine is easily determined and is a useful index in evaluating the remaining growth of the spine.

2. In the study of spinal growth, chronologic age seems to be an inadequate measure, and skeletal age should be used.

3. Growth does not cease in the spine in all instances in which the iliac apophyses complete their ossification.

4. For practical purposes, growth activity ceases in the spine at a skeletal age of 16 years, when growth rates are in the vicinity of 0.10 mm. per month.

5. Menarche is not an adequate indicator of spinal growth activity.

6. Scoliotic curves do not necessarily continue to increase while there is growth activity in the spine.

7. Scoliotic curves do not increase when the growth rate of the D8-D12 segment is under 0.30 mm. per month.

8. Progression of many scoliotic curves ceases before completion of the iliac apophyses.

9. Scoliotic curves do not increase after the skeletal age of 15 years, and in 35 per cent of the cases this skeletal age appears a year earlier than the completion of the iliac apophyses.

REFERENCES

1. Arkin, A. M.: The mechanism of structural changes in scoliosis, J. Bone & Joint Surg 31-A:519-527, 1949.
2. Cobb, J. R.: Outline for the study of scoliosis *in* Am. Acad. Orthop. Surgeons Instructional Course Lectures, vol. 5, pp 261-275, 1948.
3. Greulich, W. W., and Pyle, S I: Radiographic Atlas of Skeletal Development of the Hand and Wrist, California, Stanford Univ. Press, 1950.
4. Nachlas, I. W.: The cure of experimental scoliosis by directed growth control, J Bone & Joint Surg. 33-A:24-32, 1951.
5. Ponseti, I. V., and Friedman, B.: Prognosis in idiopathic scoliosis, J. Bone & Joint Surg 32-A:381-395, 1950.
6. Risser, J. C.: Acquired scoliosis *in* Nelson's Loose Leaf Surgery, vol. 3, pp 439-451, 1941.
7. ———: Important practical facts in the treatment of scoliosis *in* Am. Acad. Orthop Surgeons Instructional Course Lectures, vol. 5, pp. 248-260, 1948.
8. Risser, J. C., and Ferguson, A. B: Scoliosis: its prognosis, J. Bone & Joint Surg. 18: 667-670, 1936.

Observationes in Re le Crescentia del Columna Vertebral del Adolescente Feminin e in Re su Relation a Scoliosis

Summario in Interlingua

Un studio esseva interprendite pro determinar le valor de varie indicios de maturation—le etate skeletal, le crescentia del columna vertebral, le completion del ossification del apophyses iliac, le menarche—in relation al prognose de scoliosis.

Esseva studiate individuos normal e etiam individuos con scoliosis. Le valores del crescentia medie esseva determinate pro obtener un base de comparation.

Un segmenta del columna vertebral ab le octave al dece-secundo vertebra, D8 a D12, esseva seligite e le rapiditate de crescentia de iste section esseva determinate in omne casos. Ab iste valores esseva delineate curvas que esseva utile in le analyse del varie supra-mentionate indices.

Le conclusiones de iste studio es le sequentes:

1. Le rapiditate del crescentia del columna vertebral del adolescente feminin es facilemente determinabile e es un indice utile in evalutar le remanente crescentia del spina.

2. In le studio del crescentia del columna

vertebral le etate chronologic pare esser un mesura inadequate; le etate skeletal debe esser empleate.

3. Le crescentia del columna vertebral non cessa in omne casos con le completion del ossification del apophyses iliac.

4. Le activitate de crescentia in le columna vertebral practicamente cessa al etate skeletal de 16 annos, quando le rapiditate del crescentia es approximativemente 0,10 mm per mense.

5. Le menarche non es un indice adequate del activitate del crescentia del columna vertebral.

6. Le curvas scoliotic non necessarimente continua augmentar se dum le activitate de crescentia continua in le spina.

7. Le curvas scoliotic non se augmenta quando le rapiditate del crescentia del segmento D8 a D12 es infra 0,30 mm per mense.

8. Le progression de multe curvas scoliotic cessa ante le completion del apophyses iliac.

9. Le curvas scoliotic non se augmenta post le etate skeletal de 15 annos; in 35 pro cento del casos isto etate skeletal ocurreva un anno ante le completion del apophyses iliac.

5

Etiology of Spondylolisthesis*

LEON L. WILTSE, M.D.†

It is the purpose here to discuss the etiology of spondylolisthesis and to present consanguinity studies bearing upon this disease in 24 families. In these 24 families the incidence of defective neural arch at the pars interarticularis was found to be very much higher than in the general population. We shall concern ourselves only with that type of spondylolisthesis which is associated with a lesion of the pars interarticularis. Furthermore, the defect in the pars will be considered as the fundamental lesion. Whether or not there is forward slip of the anterior part of the affected vertebra, will be a secondary consideration. It is true that spondylolisthesis, when considered simply as luxation forward of one vertebra upon another, may result from a number of causes other than a defect at the pars. Examples would be severe fracture dislocation or in severe bone disease such as tuberculosis or cancer. Generally in these the etiology is fairly clear and needs no further discussion.

H. F. Kilian[8] gave us the classic description of spondylolisthesis in a paper published in 1854. One year later, Robert of Coblenz[12] established the location of the fundamental lesion in the pars interarticularis.

Over 100 years have passed since the original description by Kilian, yet the exact etiology of the lesion in the pars remains in doubt. Several theories have been advanced I will mention the more prominent ones:

1. Theory of Separate Ossification Centers. In 1859, Schwegel[16] announced that he had found in several fetuses two separate centers of ossification, one on each side of the neural arch. Therefore, he explained that failure of fusion of these two centers was the cause of the defect. This has proved to be a fallacy, because in the years since it has been well established that each half of the neural arch ossifies as one single center—Noback[11] and others—(Fig. 1). Accurate records have been kept on 509

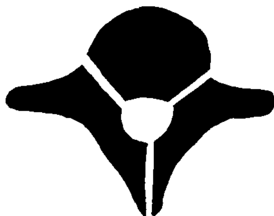


FIG. 1. Three primary ossification centers of the vertebra. Note that there is no line of fusion near the pars.

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The author wishes to thank Dr. Tom A. Kendig, ... preting the family histories from the standpoint of genetics

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FIG. 2. *(Top, left)* Spondylolisthesis following severe injury. *(Top, right)* Same case after reduction. *(Bottom)* Line defect through pars after reduction.



fetuses which have been sectioned from the sixth month of gestation to shortly after birth. *In not one single case was there evidence of* separate ossification centers, nor in fact was there any evidence even to suggest a defect at the pars interarticularis. This work was done by men whose reputation as researchers cannot be questioned (Batts,[2] Chandler,[4] Friberg,[5] Hitchcock,[7] Mall,[9] Sensing,[17] and Rowe & Roche[15]).

2. **Birth Fracture Theory.** It has been postulated that this lesion is due to a fracture at birth. Rowe and Roche[15] put this theory to rest when they experimented with stillborn infants and could not produce fracture through the pars interarticularis, either by hyperextending the spine to where the buttocks came against the upper lumbar region or by putting the symphysis pubis against the abdomen.

3. **Fracture During Postnatal Life Theory.** The fact that the incidence of defect is zero at birth (Rowe & Roche,[15] Stewart[19]) and increases to adulthood has led many to believe this to be an ordinary fracture which fails to heal. The evidence against its being an ordinary fracture is that, in review of many skeletons showing this lesion, there never is any evidence of attempted repair, whereas, in lesions in any

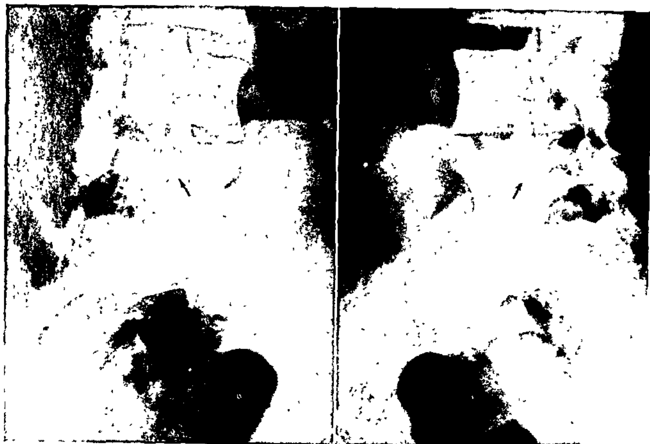


FIG. 3. Right and left pars 5 months after injury showing complete healing of both pars interarticularis.

other part of the neural arch, there is practically always evidence of attempted repair. In Stewart's[19] work on the Eskimos, he noted that whenever there was a defect in the arch other than through the pars (not including spina bifida), there was attempted healing, but never in defects through the pars.

Roche[13] reported a case in which there was known to be a fracture through the pars interarticularis, and it healed. Figures 2 and 3 show a case where there was definite fracture through the pars interarticularis with spondylolisthesis resulting, caused by an oil-well derrick having tipped over and fallen on the man. The spondylolisthesis was reduced, and the fracture healed. Thus, the only two cases of undisputed fracture of which we are aware healed.

4. Stress Fracture Theory. The idea that this is an ordinary stress fracture has been advanced by several. Here again the problem is to explain why there is no attempt at repair, since stress fractures usually have considerable callus round them. There is some evidence that constant strain may cause the pars to separate without any definite severe injury. Unander-Scharin[22] reported a case of spondylolisthesis aquisita In this case spondylolisthesis of the third lumbar vertebra was noted five years after fusion of the third, the fourth and the fifth lumbar vertebrae to the sacrum. Previous roentgenograms had revealed no evidence of lesion. This would suggest that the added strain on the pars of having the lamina and the inferior facets of L-3 solid to the vertebrae below, and all the weight of the body above, caused the pars to separate. Anderson[1] also reported a case of spondylolysis of L-4 when L-5 had been fused to the sacrum. In this case, of course, the element of strain



FIG. 4 (All 3 illustrations are of the same case). *(Top, left)* Spondylolisthesis in a case of unilateral defect. *(Top, right)* Defective pars on left. *(Bottom)* Elongated and attenuated, but intact, pars on the right. This patient underwent surgery, at which time it was noted that the posterior element was solid, thus proving the defect to be unilateral.

is not as precise, because the vertebra in which the lesion occurred was free. There are cases of unilateral defect along with forward slip of the vertebrae where the intact pars is longer than it should be, as compared with the vertebra above. This would suggest that the pars on the intact side tended to crack through and heal in an elongated position (Fig. 4) Occasionally, we see forward slip of a vertebra with both pars intact, but elongated and often attenuated (Fig. 5).

5. Weakness of Supporting Structure

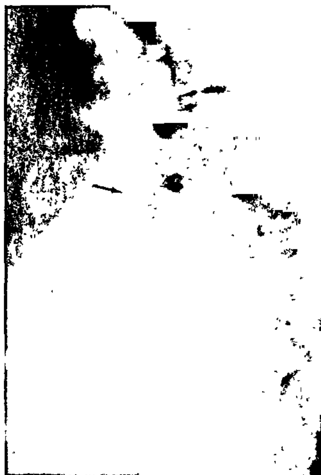


FIG 5. Spondylolisthesis with both pars intact.

Theory. P. H. Newman[10] has advanced the theory that the characteristic lesion in the pars interarticularis is secondary to instability in the low back. He believes that acquired deficiency of the lumbodorsal fascia, other posterior spinal ligaments and the intervertebral disk contribute to this instability. The pars defect, he says, is not the cause of spondylolisthesis but is a secondary result of instability. Presumably the instability puts extra stress on the pars, so that it breaks. He believes that originally there was nothing wrong with the pars. Against P. H. Newman's theory must be considered the following:

(A) We often see defective pars in young children who seem to be perfectly well formed, have goo[illegible] posture and have none of the so-called t[illegible]al build of the person with spondylolisthesis. It is difficult to see how the factor of extra strain or weakness is at work here.

(B) Women have a much lower incidence of defect than men, yet women have more relaxed ligaments than men and more of a tendency toward lumbar lordosis. Bosworth *et al.*[3] found that as regards the amount of slipping of the affected vertebra in the average female with spondylolisthesis, the vertebra had slipped forward 25 per cent, whereas in the average male it had slipped 14 per cent in their series. They suggested that this might be due to less efficient ligamentous, muscular and bony supporting structures in the female.

(C) In cases where the back muscles have been paralyzed by polio, defects have not been noted to develop more often than average. It would seem that these polio victims might develop defects due to the fact that the muscles are not doing their work and more strain goes on the bony parts.

(D) Pars defects occur occasionally in parts of the spine where there is very little strain. Stewart[20] found the incidence in other parts of the skeleton than the lower lumbar to be fairly high.

(E) Friberg[5] reported the case of an 11-month-old infant which had spondylolisthesis of five vertebrae. The child never had sat up, nor had it been exposed to trauma. There never was any possibility of strain in this child.

6. **Pathology at Pars.** The idea that there is aseptic necrosis, laking of blood or cyst formation in the pars has been advanced, but on microscopic examination of many specimens no evidence of such can be found.

7. **Lumbar Lordosis.** Lumbar lordosis as a precipitating factor has been studied extensively by Stewart[21] in the Eskimo skeleton. According to his findings, there does seem to be some relationship between lumbar lordosis and spondylolisthesis, although

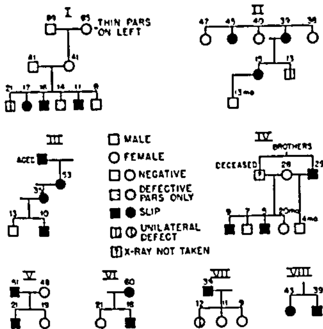


FIG. 6. Eight of the families studied are shown above. Roentgenograms of two of the families, I and III, are presented.

Bosworth *et al.* could not make such a correlation in their studies.

NATURE OF THE LESION

The defect itself has been reported upon by a number of observers. Gill[6] believes that the defect is most commonly filled with a mass of fibrocartilaginous tissue. Roche[14] found the ligaments bridging the defect to be heavier than average but no evidence of callus formation or anything that would suggest a fracture.

Bosworth, *et al.*[3] found the following four different types of situation at the defect:

1. Thin, tenuous, fibrous bands bridging the defect
2. Thick and heavy fibrous columns in the same location
3. A bony bridge across the portion of the arch where a defect had been suspected
4. A false joint

Combinations of these such as fibrous attachment and pseudojoint on the other side were also found.





FIG 7. *(Top)* Line defect in pars of oldest (age 21) member of Family I. *(Bottom)* Severe spondylolisthesis in 17-year-old female member of Family I.

We studied the defect grossly and microscopically in six cases where the posterior element had been removed at surgery. Great care was taken to orient the sections so as to follow the transition from bone out into the defect. In no case was there endochondral or intramembranous ossification. No osteoid or cartilaginous callus was seen, nor was there evidence of cystic change. There was no periosteum over the end of the bone at the defect. There was no evidence of attempted healing. One outstanding finding

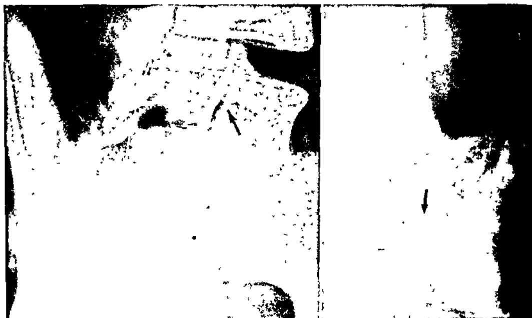


FIG. 8. *(Left)* Severe spondylolisthesis in 16-year-old boy of Family I. *(Right)* Definite pars defect in 14-year-old boy of Family I.

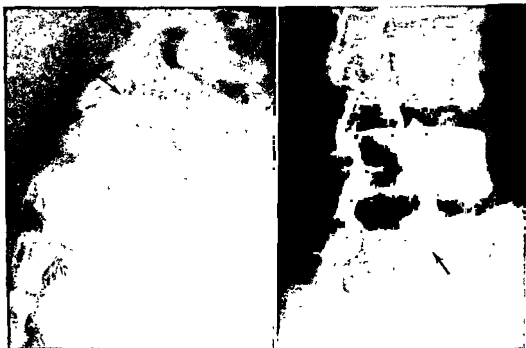


FIG 9 *(Left)* Severe spondylolisthesis in 11-year-old boy of Family I. *(Right)* Only negative member of the six children of Family I.

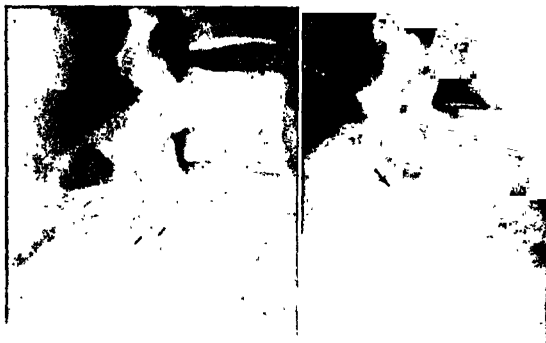


FIG. 10. *(Top, left)* Female, age 53, second generation of Family III, shows spondylolisthesis. *(Top, right)* Female, age 35, third generation of Family III, shows spondylolisthesis. *(Bottom)* Male, age 10, fourth generation of Family III, shows definite defects with mild slip.

was finely fibrillar fibrous connective tissue completely acellular extending right up to the bony cortex at the defect.

PRESENTATION OF NEW MATERIAL

In 1950 a 15-year-old boy who had severe spondylolisthesis was seen in the office. The family requested that roentgenograms of the other children in the family be taken. There were six children, five of whom had defects at the pars interarticularis. This aroused my interest in the possible familial aspect of this condition; therefore, during the next few years roentgenograms were taken of ten more families. In these first 11 families the incidence was very much higher than the usual incidence of 5 per cent. We then went through the cross index and were able to get 13 more families to submit to the taking of roentgenograms, thus making a total of 24 families. The only families chosen were those in which there was definite spondylolisthesis with forward slip in the patient who originally had come to the office. We did



not roentgenograph families in which there was only spondylolysis in the original patient, because we wanted to be absolutely sure that there were defects in the original patient. These families were called in routinely by the x-ray technician. The majority could not be located or, for one reason or another, could not bring their families in for roentgenograms. In those families studied, of course, we could not get all the members either, but included in the study are all members who did come in. Thus, from a statistical standpoint, we believe it to be accurate. There is one factor which should be mentioned; that is, that when families were contacted routinely without a great deal of special explanation of what the study was all about, there was a tendency to bring in only the younger members. This happened because they were the ones about whom there was concern. This tended to weight the statistics with younger members, and, as has been noted before, the incidence of the defect is low in children and increases up to adulthood.

Six views were taken routinely—large anteroposterior and lateral, spot lateral right and left lateral obliques, and 20° anterior oblique views of the lumbosacral area.

In collecting the statistics, only siblings, direct descendants or direct forebears were included. Of these 24 families, 67 relatives were roentgenographed. The initial patient of each family who prompted the study of that family was not included in the statistics because he was selected.

Figure 6 shows consanguinity studies of eight of these families. Figures 7 to 9 show roentgenograms of the five positive members of Family I, and Figure 9, right, shows the one negative member; incidentally, the youngest, age 8.

Figure 10 shows the roentgenograms of the positive members of Family III. Only one of this family was negative, a 13-year-old boy. The roentgenograms of the oldest member were not available, but his records were; a spinal fusion for spondylolisthesis had been performed on him over 20 years before.

Age and sex distribution of the 21 relatives showing defects is shown in Figure 11.

STATISTICS FROM OUR STUDIES

Total Incidence

Families	Relatives Roentgenographed	Defects	Incidence
24	67	21	31.3+%

Incidence As to Sex

		Positive	Incidence
Males roentgenographed	33	14	42.1%
Females roentgenographed	34	8	23.5%

Incidence of Slip As to Sex

	Total defects	Slip	Incidence
Males	14	8	57.1%
Females	8	6	75.0%

DISTRIBUTION AS TO AGE AND SEX

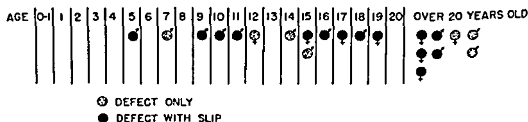


Fig 11 Note the paucity in younger members. The incidence hits its stride at about age 10 and continues to adulthood in this study.

Incidence in Cases Under 20 Compared With Those Over 20

		Defects	*Incidence*
No. under 20	42	13	30.9%
No. over 20	25	8	32.0%

Incidence of Unilateral Defects

		Unilateral Defects	*Incidence*
Total number	21	5	23.8%

Figure 12 shows a further study of Family I. It is to be noted that the mother in the first generation has a thin attenuated pars. Then her daughter is negative as well as the daughter's husband in the second generation. All members of the husband's family of that second generation are negative, but his children are positive in the ratio of five to one. This suggests that the mother in the second generation is a carrier of the defect.

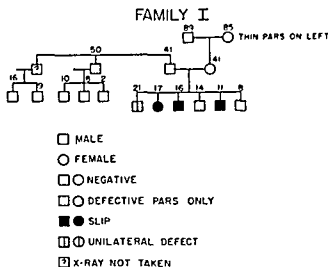


Fig. 12. Note that the female in the first generation has a thin attenuated pars. Her daughter is negative as well as the [illegible] ratio of 5 to 1. This suggests that the mother in the second generation is a carrier of the defect.

CONSANGUINITY STUDIES

Our material, along with all the available material in published reports, was subjected to consanguinity studies to try to determine whether the trait is dominant or recessive. Dr. Philip Hildreth, of Long Beach State College, assisted in this phase of the work. It was found to be impossible to determine definitely which it is The greatest weight of evidence is on the side of its being due to a recessive gene. If it is recessive, then about 34 per cent of our population are carriers of the trait and are heterozygous. If a heterozygous person marries a normal, then the offspring will be affected at the rate of 25 per cent. If two heterozygotes marry, then this chance is 50 per cent. The chances of two heterozygotes marrying are about 34 per cent in our population, when the total incidence in the general population is 5 per cent.

Unilateral defects and variation in the amount of slip in the involved vertebra indicate that the trait is capable of different expression. It is not sex linked.

Sorsby[18] in his book *Clinical Genetics* cites the extensive work of T. D. Stewart[21] on Eskimos. It was felt by him that the trait in both minor and severe degrees was the result of gene influence, most likely dominant, with varying degrees of expressivity, but with good penetrance.

A separate study is being conducted on this question of gene dominance and will be reported in the next year.

DISCUSSION

In cases studied, only siblings, direct forebears or direct descendants were included in the statistics. The patient who came originally was not included as he was selected in that he came to the office because of backache. It is believed that, except for the fact of being related to a person with spondylolisthesis, these relatives simply represent a segment of population, and, if no familial factor were present, the incidence of defective pars would run around 5 per cent.

It should be noted that in families in which the incidence of lesion is running very high, it is in the youngest members that no defect can be detected. It is the plan to wait

about 10 years, then call in these younger members who now are negative and see how many have become positive. We now have good roentgenograms of a rather large number of young children from families with a high incidence of lesion. If it can be proved that a fair number of these previously negative children have become positive, we shall have gone a step farther in discovering the origin of this baffling lesion.

Before postulating on the etiology of this condition, let us review some of the established facts about this disease.

1. No evidence of defect at birth
2. Incidence of defect increases to adulthood in Caucasians and Negroes, and to 34 in Eskimos.
3. Very scant roentgenographic evidence of the defect under 4 or 5 years of age. Our youngest was 5; Hitchcock had a case 4; and Friberg had one 11 months.
4. The incidence of defect varies considerably in different races. It runs as high as 50 per cent in some Eskimos. Rowe and Roche quote the incidence in Americans as white male, 6.4 per cent; Negro male, 2.8 per cent; white female, 2.3 per cent; and Negro female, 1.1 per cent. Orientals range about 3.5 per cent to 4 per cent.
5. The only unquestionable pars fractures we know of have healed.
6. Defects in other parts of the neural arch attempt to heal, but there is practically no attempt at healing of the pars.
7. Only rarely does the amount of slip increase. Apparently, slip occurs shortly after the appearance of the defect.
8. No evidence of cystic condition, aseptic necrosis or laking of blood in pars from studies so far.
9. From our studies the disease would appear to be familial.

Taking all the above facts into account, the following concept of etiology has been formulated: the defect through the pars interarticularis is due to a congenital weakness or dysplasia at this point Due to the particular stresses upon this segment of the spine, gradual lysis occurs. Occasionally, instead of complete lysis, healing takes place which accounts for the elongated pars in some cases and may account for the unilateral defect (other cases of unilateral defect may be dysplastic only on one side). Due to the peculiar construction of this area, attempt at repair with callus formation does not occur as it does in other parts of the body. A certain congenital element is necessary or this fracture would not occur from simple stresses, or, if it did occur from severe trauma, it would heal as it did in the two cases cited. The exact nature of this "congenital weakness" is unknown.

CONCLUSION

Consanguinity studies were done on 24 families in which the initial patient had a defective pars interarticularis. Only direct forebears, siblings and direct descendants were included in the statistics, but all of these in all the families were. The original patient never was included because he was selected. In these relatives, the incidence of defective pars was 31.3+ per cent as compared with 5 per cent in the general population.

From these studies it would appear that the lesion in the pars results from dissolution of continuity of the bone due to a congenital weakness at this point. It cannot be said definitely from these studies whether the trait is dominant or recessive, but the greatest weight of evidence is on the side of its being recessive. It can be stated that it is not sex linked and has varying expressivity. The exact nature of this "congenital weakness," which permits this breakthrough or dissolution to occur, remains an enigma

REFERENCES

1. Anderson, Carl E.: Spondyloschisis following spine fusion, J. Bone & Joint Surg. 38-A: 1142-1146, 1956.
2. Batts, Martin, Jr.: The etiology of spondylolisthesis, J. Bone & Joint Surg. 21:879-884, 1939
3. Bosworth, David M., Fielding, William,

Damerest, Lawrence, and Bonoquist, M.: Spondylolisthesis: a critical review of a consecutive series treated by arthrodesis, J. Bone & Joint Surg. 37-A:767-786, 1955.
4. Chandler, F. A.: Lesions of the "isthmus" (pars interarticularis) of the laminae of the lower lumbar vertebrae and their relation to spondylolisthesis, Surg., Gynec. & Obst. 53:273-306, 1931.
5. [illegible]
6. Gill, Gerold G., Manning, John G., and White, Hugh L.: Surgical treatment of spondylolisthesis without spine fusion, J. Bone & Joint Surg. 37-A:493-520, 1955.
7. Hitchcock, H. H.: Spondylolisthesis; observations on its development, progression, and genesis, J. Bone & Joint Surg. 22:1-6, 1940.
8. Kilian, H. F.: De Spondylolisthesi gravissime Pelvangustiae caussa nuper detecta, Commentatio anatomico-obstetrica, Bonnae, Lit c. Georgii, 1854.
9. Mall, F. P.: On ossification centers in human embryos less than 100 days old, Am. J. Anat 5:433-458, 1905.
10. Newman, P. H.: Spondylolisthesis, its cause and effect, Ann. Roy. Coll. Surgeons England, July-June, 304-322, 1954-1955.
11. Noback, C. R.: Developmental anatomy of human osseous skeleton during embryonic, fetal, and circumnatal periods, Anat. Rec. 88:91-125, 1944.
12. Robert (zu Coblenz): Eine eigenthümliche angeborene Lordose, wahrscheinlich bedingt durch eine Verschiebung des Körpers des letzten Lendenwirbels auf die vordere Fläche des ersten Kreuzbeinwirbels (Spondylolisthesis Kilian), nebst Bemerkungen über die Mechanik dieser Beckenformation, Monatsschr. Geburtskunde u. Frauenkrank. 5:81-94, 1855.
13. Roche, M. B.: Bilateral fracture of the pars interarticularis of a lumbar neural arch, J. Bone & Joint Surg. 30-A:1005-1008, 1948.
14. ———: The pathology of neural arch defects, a dissection study, J. Bone & Joint Surg. 31-A:529-537, 1949.
15. Rowe, G. G., and Roche, M. B.: Etiology of separate neural arch, J. Bone & Joint Surg. 35-A:102-110, 1953.
16. Schwegel, A.: Über knochenvarietäten, Ztschr. rationelle Med. 5:283-319, 1859.
17. Sensing, E. C.: The early development of the human vertebral column, contributions to embryology 33:21-42, Carnegie Inst. of Washington, 1949.
18. Sorsby, Arnold: Clinical Genetics, St. Louis, Mosby, 1953.
19. Stewart, T. D.: The age incidence of neural arch defects in Alaskan natives considered from the standpoint of etiology, J. Bone & Joint Surg. 35-A:937-950, 1953.
20. ———: Incidence of separate neural arch in the lumbar vertebrae of Eskimos, Am. J. Phys. Anthropol. 16:1, 1931.
21. ———: Personal communication.
22. Unander-Scharin, Lars: A case of spondylolisthesis lumbalis aquisita, Acta orthop. scandinav. 19:536-544, 1950.

Etiologia de Spondylolisthese

Summario in Interlingua

Studios de consanguineitate in re iste morbo esseva executate in 24 familias. Esseva constatate que le incidentia de defectos del arco neural al parte interarcticular esseva multo plus alte que in le population general.

In sex casos, le defecto in le parte interarticular esseva studiate macro- e microscopicamente. Nulle signos de ossification endochondral o intramembranose esseva notate. Periosteo non se presentava supra le termino del osso al sito del defecto. Nulle manifestation de un processo de curation esseva notate. Un constatation eminente esseva tessuto conjunctive fibrose fibrillar, que esseva acellular e se extendeva al cotice ossose del defecto.

Vinti-quatro familias esseva subjicite a studios radiologic e genealogic. Solmente ancestres directe, fratres e sorores, e descendentes directe esseva includite in le statisticas. Le patiente original esseva semper excludite, proque su caso representava ab le puncto de vista statistic un caso seligite. In le gruppo total del consanguineos le inci-

dentia de defectos amontava a 31,3 pro cento. Le valor correspondente in le population general es 5 pro cento.

Il non esseva possibile determinar con certitude si le tracto es dominante o recessive, sed le peso comprobatori del datos colligite favora le conclusion que illo es recessive. Un studio separate es in progresso pro clarificar iste puncto. Illo va esser reportate le anno proxime. Defectos unilateral e variationes in le grado del dislocation indica que le tracto es capace de expressiones differente. Illo non es ligate al sexo.

Super le base de iste studios, le autor formula le sequente concepto del etiologia: Le defecto a transverso le parte interarticular es le resultato de un debilitate o un dysplasia congenite a iste puncto. A causa del stress particular a que iste segmento del columna vertebral es exponite, il occurre un lyse gradual. A vices il occurre in loco del lyse complete un processo de curation. Isto explica le elongation del parte que es incontrate in certe casos. Possibilemente illo etiam explica le occurrentia de defectos unilateral. In consequentia del construction peculiar de iste area, essayos de reparo con formation de callo non occurre, per contrasto con lo que es le norma in altere portiones del corpore. Il es necessari supponer un certe elemento congenite. Sin un tal, iste fractura non occurrerea in consequentia de simple stresses. E si illo occurreva in consequentia de trauma sever, illo se curarea. Le natura exacte de iste "debilitate congenite" remane un enigma.

6

Legg-Calvé-Perthes Disease—Results of Treatment

Jacob F. Katz, M.D.*

BACKGROUND

Legg-Calvé-Perthes disease remains the same etiologic enigma since its recognition as a clinical entity in the classic descriptions of Legg,[11] Perthes[15] and Calvé.[2] The gamut of causative relationships has run from tuberculosis, with which it was classified confusedly at first, to less specific inflammations, metabolic abnormalities, vascular inadequacies, idiopathic or hereditary predilections, hormonal imbalances[10] and injury. Each of these theoretical possibilities has had its champion at one time or another, but none has received general acceptance.

From a teleologic point of view there is agreement that the mechanism of the Perthes change is consistent with avascular necrosis. This was ably shown by Lippman[19] in his animal experimentations, in which he created a Perthes-like process in the femoral heads of rabbits after ligating the ligamentum teres. Confirmation by him subsequently was possible on human material,[13] when femoral heads in Legg-Perthes disease were made available because of reconstructive surgical procedures then performed. Using needle biopsy[9,16] and curettage[6] technics, many others also have demonstrated since a presence of noninfected dead bone in the proximal femoral epiphysis. Observations of the similarity of Perthes disease to that of known femoral head vascular loss in extensively slipped femoral capital epiphyses and femoral neck fractures provided suggestive collateral confirmation.

For the past decade the emphasis in the literature has shifted to the evaluation of therapeutic modalities. Unfortunately, no greater agreement existed here than in the etiologic studies. Wide disagreement was found to exist. This ranged from a nihilistic point of view which questioned the value of any treatment to the extreme conservative viewpoint which required strict, systematic immobilization for prolonged periods of time. In a long-term analysis of 153 cases, Sundt[18] concluded that "measures of treatment failed to prevent the occurrence of the secondary arthrosis which is the cause of the trouble that follows in the wake of the disease." As far back as 1927, Legg,[12] reporting on the end-results of coxa plana, stated that "relief from weight bearing has in no way affected the end result."

That this point of view was not generally accepted was well emphasized by those who discussed Legg's paper. Dr. F. C. Kidner, in commenting, stated, "I do not agree with Dr. Legg that protection is not necessary." In his discussion at the same time, E. W. Ryerson stated that "weight bearing should not be permitted in cases of Legg's disease, because the femoral head is softened and

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will become flattened if allowed to press against the acetabulum."

A survey of the world literature on the therapy of Legg-Calvé-Perthes disease only confirmed the tug of war that has been going on in battling the problem of therapy. That Sundt cannot be accepted as the Scandinavian spokesman is emphasized by a recent report coming out of Norway, by Hauge,[5] who found after investigating 100 patients that results with 1 year's bed rest were "better than those of non-treated patients, if weight bearing ceases early in the disease—about 60% against 25%."

Since the integrity and the caliber of the many investigators engaged in Perthes research cannot be impugned, one must seek the explanation of the contrary viewpoints elsewhere. Certain deficiencies are obvious in an examination of the various study reports. One of these is the matter of treatment which, at times, is very loosely interpreted. Sundt's series, for example, included patients kept in bed from 1 to 4 weeks as treated cases He recognized this defect but felt that the expediency of his study made the division between treated and nontreated cases an arbitrary and necessary one. Another factor of significance is the stage of disease at which treatment first is started. Many investigators have taken this into consideration. A third important defect lies in the multiplicity of methods of evaluating the results of therapy. It is unusual to find two reports in which similar criteria for measurement of end-results have been used. It has been a common experience for authors to develop rather arbitrary standards to suit their own purposes, thus making it difficult to translate their experience to that of others. This lack of standardization and establishment of criteria by which one series of cases can be compared with another represents the greatest difficulty. Only when bases for comparison have been drawn meticulously and utilized by all workers can some resolution of the present existing confusion come about.

METHODS UTILIZED FOR THE CALCULATION OF END-RESULT

Sundt developed four categories. He labeled these C F I, C F II, C F III and C F IV to represent the approximate shape of the head at the termination of the disease The Roman numeral I indicated a spherical head; Roman numeral II, an ovoid; Roman numeral III, a cylindrical head; and Roman numeral IV, a quadrangular-shaped head For the end-result, which did not clearly fall into any of these four categories, he created combinations so that a given result could be labeled either II (III) or III (IV) or other combinations, depending on the many possible transitions. This was clearly a subjective type of analysis, and in the hands of a careful observer undoubtedly merited consideration. Its limitation to duplication by others is self-evident.

Hauge, in his study, devised three categories for his analysis: a ball-shaped epiphysis *as one representing a good* result; a flattened, but evenly rounded, epiphysis as a *fair* result; and an angular epiphysis as a *poor* result.

Jonsater suggested tentatively an index which he called the Caput Index, based on the relationship between the height and the width of the shadow cast by a diodrast hip arthrogram. Since this required highly specialized study technics, not usual in any routine, it had limited application

Goff[4] favored a description evaluation for his end-results in a manner resembling Sundt's classification. He also suggested a separation into spherical, mushroom and malum coxae juvenilis types, each of these major groupings having subdivisions of different severity.

Eyre-Brook[3] suggested the use of an Epiphyseal Index, which he felt would satisfy the requirement of statistical analysis. It represented the relation of the height of the regenerated epiphysis to its width multiplied by 100. It was a measurable mathematical entity and could be duplicated. In his own material he found that a reliable relationship

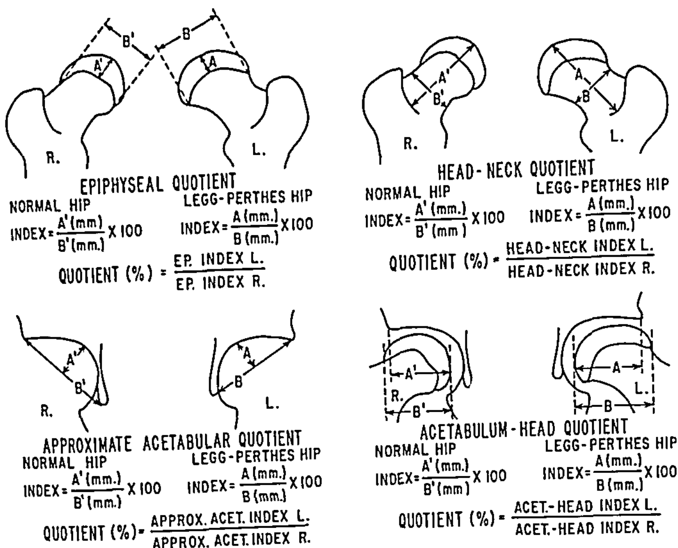


FIG. 1 (After Heyman & Herndon: J. Bone & Joint Surg. 32-A:767-778)

existed in the epiphyseal indices of children; those under 7 years of age normally had an index of from 45 to 55, while those over 7 years of age fell in a range between 35 and 45. In an analysis of his own cases treated in recumbency in traction, using this index he found that "the results are definitely superior to those obtained by other methods."

Sjovall[17] expressed the residual deformity in Legg-Calvé-Perthes disease as the quotient of the epiphyseal index of the involved side, as determined by Eyre-Brook, with the epiphyseal index of the hip on the uninvolved side.

Heyman and Herndon[7] proposed making their determination more inclusive by taking into account in the final determination the composite influence of the shape of the head, the neck and the acetabulum as a numerically recorded comprehensive quotient. In Figure 1 I have redrawn from their publication the details involved in the measurement of these many quotients, which, when averaged together, resulted in a comprehensive quotient.

Thus we see that the problem of determining suitable means of communicating the end-results has stimulated considerable thought, but the solution has been varied and uncertain. It is evident that the translation of the results from one set of material, using a given criterion, is extremely difficult if the results of another are expressed differently.

As a device for measuring accurately the end-result in Legg-Calvé-Perthes disease, the method devised by Heyman and Herndon seemed to be most impressive. All the cases

under analysis in this study were examined, using the method leading to a comprehensive quotient. When this rather extensive calculation was completed, it became apparent that, despite the claims of the originators, a striking relationship existed between the epiphyseal quotient and the final comprehensive quotient. Figure 2 shows the practically linear relationship which was present when the epiphyseal quotient was plotted against the comprehensive quotient. Possibly this could have been anticipated, since there has been general agreement that the major component of the final result is contributed by the perfectness of the reconstitution or degree of deformity of the femoral capital epiphysis. In view of the demonstrable close relationship between the epiphyseal quotient and the more difficultly determined comprehensive quotient, the subsequent major statistical analysis of the cases in this report was computed on the basis of the epiphyseal quotient alone. As mentioned earlier, this consisted of the ratio of the epiphyseal index of the involved side to the epiphyseal index of the uninvolved side. Utilizing this method, bilateral cases could not be compared. It is because of this objection that Goff had questioned the universality of such mathematical treatment.

MECHANICS OF STUDY

All the children included in this report were treated at Blythedale. This institution affords the opportunity for protracted inhospital stay while at the same time offering a country, convalescent setting with available formal schooling, physical therapy, medical supervision, group and recreation therapy, social service and psychiatric supervision.

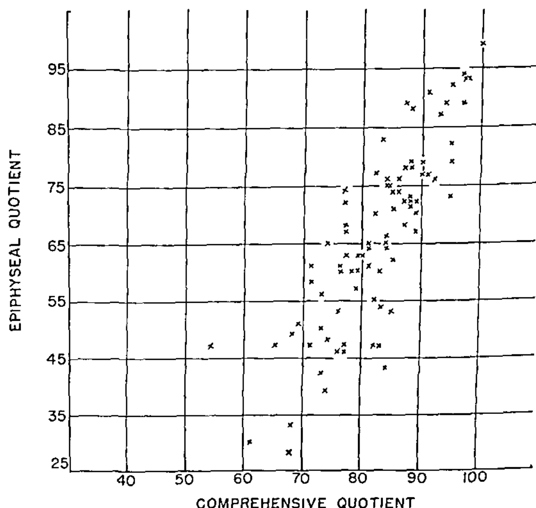


FIG. 2 See text.

The patients come from many different sources, mostly from the large hospitals of Metropolitan New York. Recommendations for their therapy, in general outline, usually come with the patients. In prior years, prescription for therapy included bed rest, alternating with other compromise therapies, such as wheel-chair, crutch or brace ambulation. In the past 8 years, because of my interest in this disease, the direction of



FIG. 3. These photographs demonstrate the traction device, which consists of shoes attached to a crossbar.

therapy was slanted specifically toward prolonged continuous bed rest in order to test the thesis that Perthes end-results could be influenced by such discipline. In addition to bed rest, a device was used which consisted of shoes fastened firmly to a wooden crossbar through which a small amount of traction, 3 to 5 lbs., was applied to the lower extremities of the children. Such a small amount of weight could only elicit light traction, and it is for that reason that it could be applied safely through shoes. No claim is made that the traction diminished effectively much of the muscle pull normally serving to hold the upper femur to the pelvis. It was felt, rather, that the traction apparatus served to make the enforcement of bed rest a little easier while contributing minimal pull. Even under this arrangement, meticulous foot care was necessary to ensure that no skin pressure areas developed. That no significant complication in this direction was ever any problem was due to excellent nursing supervision. Figure 3 demonstrates the shoe-traction apparatus. As will be seen, this device permitted children to sit in bed during the day and to allow active hip motion. This arrangement did not interfere with the molding between the acetabulum and the femoral head that inevitably took place in this controlled situation. In addition to the active movement possible in bed within the limitations of traction, a short specifically prescribed period of formal active non-weight-bearing exercise was conducted under physiotherapeutic guidance daily.

An additional group of patients now are being treated concurrently at Blythedale; they are immobilized in a metallic frame which similarly permits hip movement but selectively places the limbs in abduction without traction. The results of therapy with this variation are *not* included here.

In this study, we decided upon the following arbitrary subdivisions of the epiphyseal quotient, which followed approximately the suggestions of Herndon and Heyman for their comprehensive quotient:

A *good* result was one with an epiphyseal quotient between 75 and 100; a *fair* result, from 50 to 75; a *poor* result, from 35 to 50; and a *bad* result, from 0 to 35. The combination of the *fair* and the *good* groups we considered to yield a satisfactory result. The *poor* and the *bad* categories were deemed to be unsatisfactory. Although some of the cases included in this study were completed many years ago, and afforded us reasonably good long-term follow-up, it was not the point of this chapter to conduct a long-term follow-up study of the end-result. By specific choice the limitation set for this study was that of judging the roentgenographic result of the femoral capital epiphysis at the termination of therapy when stabilization occurred in the femoral head changes.

It has been stated many times in the literature that the roentgenographic result is not necessarily consistent with the clinical result. However, the margin of deviation is such that the clinical picture is usually better than the roentgenographic evidence would indicate. By making the x-ray the basis for the present evaluation, the study was definitely only an interim one. However, the experience obtained from our limited long-term follow-up was such that we were convinced that our satisfactory results would stand up under the test of time. The femoral capital epiphyses of minimal deformity, as indicated by the epiphyseal quotient, presented good joint congruity and a well-restored femoral epiphysis, and were least likely to develop major secondary arthritis in later life.

As will be evident from a study of the reproductions of the roentgenograms, the descriptive categories of *spherical, ovoid, cylindrical* and *quadrangular* or other methods can be identified with the numerical ratings adopted here. It should be mentioned that one or two of the cases included in this study had had drilling, surgical operations (Fig. 4) on the neck and the epiphysis, but the results were not convinc-



FIG. 4 G. W. Age at onset, 4 years Symptoms started with right-sided limp and pain following fall out of bed in July, 1938. Limp disappeared until another fall, in April, 1939, when it returned. Roentgenograms were positive for Perthes' disease. In May, 1939, patient had drilling of the femoral head and plaster cast application. She was admitted to Blythedale in June, 1939, where the plaster cast was kept on until July, 1940. Thereafter she was fitted with a caliper brace until January, 1941, when she was discharged.

On the last follow-up visit on November 6, 1956, the patient noted occasional pain in the right hip in damp weather but was able to do everything. There was ⅝-inch shortening of the right leg without atrophy. There was a 10° loss in abduction and a 15° loss in internal rotation

The epiphyseal quotient in 1940 was 64 and in 1956 was 68. The hip has held up reasonably well The only roentgenographic change of note is the small lateral prominence of the acetabulum on the right, suggesting osteophyte formation.

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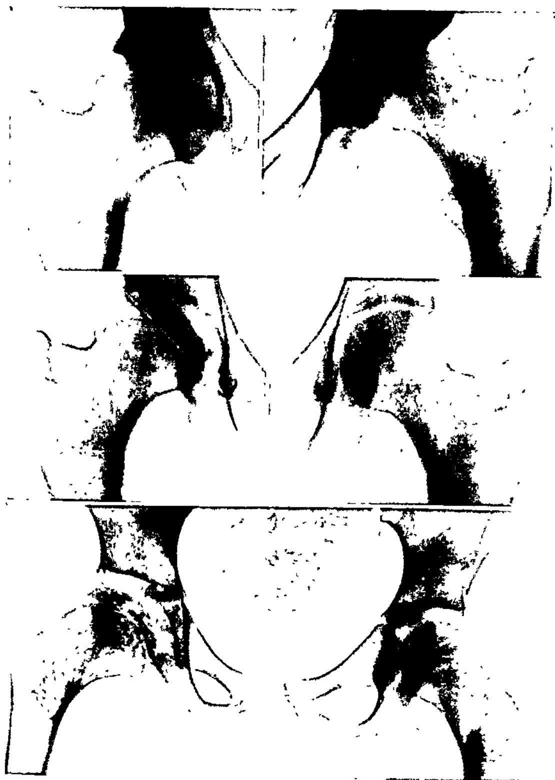


FIG. 4. G. W. Age at onset, 4 years. Symptoms started with right-sided limp and pain following fall out of bed in July, 1938. Limp disappeared until another fall, in April, 1939, when it returned. Roentgenograms were positive for Perthes' disease. In May, 1939, patient had drilling of the femoral head and plaster cast application She was admitted to Blythedale in June, 1939, where the plaster cast was kept on until July, 1940. Thereafter she was fitted with a caliper brace until January, 1941, when she was discharged.

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FIG. 5. D. B. Age at onset, 4½ years. However, treatment was not commenced until 1 year later, in April, 1950. This consisted of crutches and a sling, until February, 1951. Then a plaster cast was applied. This was kept on until the patient's transfer to Blythedale in June, 1951. He was kept in bed with traction until March, 1952, and then on crutches until discharged in November, 1952. Crutches were continued until January, 1953, after which he walked without restriction. Treatment here was erratic and included plaster cast fixation. Epiphyseal quotient—47.





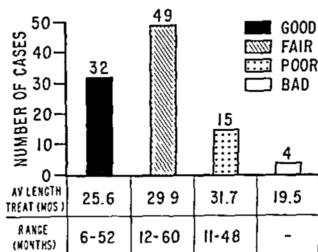


FIG. 6 See text

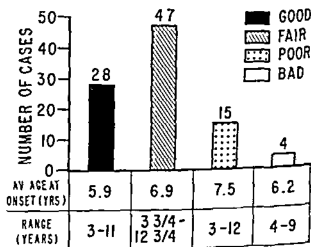


FIG. 7. See text.

ingly different from those in which no such procedure was performed. The majority of our cases were treated purely conservatively with restriction of weight-bearing. Bozsan[1] and Howorth[8] especially had experience in the surgical adjuvant in the therapy of Perthes' disease, but this has not gained wide compliance. A few of our patients were also treated in plaster casts (Fig. 5) while resting in bed, and, since these also were found to contribute no advantage over those treated more conventionally, these were not regarded differently in the statistical analysis.

RESULTS OF STUDY

One hundred and eleven cases formed the basis of this study. Eleven of these cases were bilateral with major involvements in both hips and were excluded from this analysis, leaving 100 cases of unilateral epiphyseal involvement. Ninety of these patients were males and 21 were females. No significant localization between the left hip and the right hip was found. The average age for the entire group was 6.7 years, with a range extending from 3 years to 12 years and 9 months, indicating that the disease occurred preponderantly in the younger child.

Figure 6 shows the breakdown of our material according to epiphyseal quotient. The 32 *good* and the 49 *fair* cases contributed 81 per cent satisfactory results. The 15 *poor* and the 4 *bad* cases made up 19 per cent unsatisfactory results. Relating those categories to total non-weight-bearing treatment, including all forms of relief of weight-bearing, it was found that the 32 *good* cases were treated an average length of 25.6 months. The average length of treatment, including all forms of therapy, for the 49 *fair* cases was 29.9 months. In the 15 *poor* cases, the average length of total treatment was 31.7 months In the 4 *bad* cases, the average length of treatment was 19 5 months. In this last category, the explanation for the shortened, total *treatment* lay in the fact that treatment essentially had

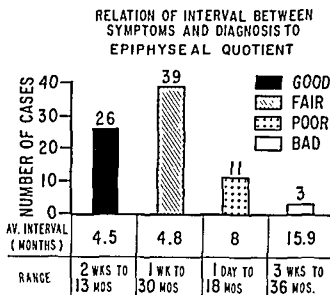


Fig. 8. See text.

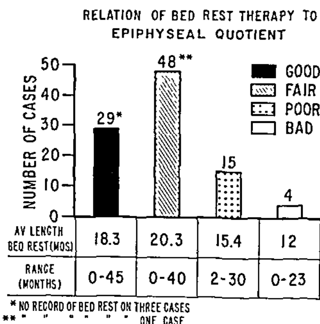


Fig. 9. See text.

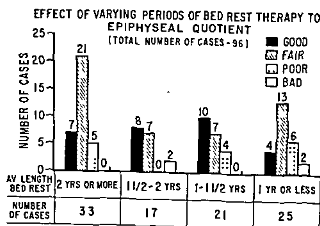


Fig. 10. See text

started late and was continued for less additional time.

Sundt stated it "to be a general experience that the younger the patient is, the better is the prognosis in this disease." Herndon and Heyman stated that the only factor of real, prognostic importance was the age of the patient. They found that, with few exceptions, the younger the patient the better the result. In Figure 7, in the 28 *good* cases in which the information was available, the average age at onset was found to be 5.9 years, with the range extending from 3 to 11 years. In the *fair* category, in the 47 cases, where the information was obtainable, the average age at onset was 6.9 years, the range being 3 years and 9 months to 12 years and 9 months. In the 15 *poor* cases, the average age at onset was 7.5 years, and in the 4 *bad* cases, the average age at onset was 6.2 years. Although the discrepancy in ages was not overwhelming, the trend indicated that the younger the child the better the prognosis and agreed with the consensus in the literature.

The duration of the disease prior to the onset of therapy has been held to be an important factor in the prognosis. Mindell and Sherman[14] in their study of the late results in Legg-Perthes disease found that patients should be treated early. Hauge also found in his study that the number of good and fair results increased considerably if treatment commenced early. In Figure 8 we have shown graphically the distribution of our results according to the interval between symptoms and diagnosis. In the *good* category, where the information was available in 26 cases, the average interval between symptoms and diagnosis was 4.5 months, the range being from 2 weeks to 13 months In the 39 *fair* cases in which this information was available, the average interval was 4 8 months. In the *poor* and the *bad* categories, the average intervals were 8 and 15 9 months, respectively. Although the ranges indicate wide variability, the figures suggest rather definitely that the earlier the disease is treated the better is the end-result.

The final facet which should alter the prognosis and the end-result in Legg-Calvé-Perthes disease is the efficacy of the therapy. We have already indicated that 81 per cent of our cases were satisfactory, as judged by the epiphyseal quotient at the termination of treatment. In analyzing the facts to determine what part bed rest played in the satisfactory group of cases, we find in Figure 9 that of the *good* cases in which information was available in 29, the average time spent on bed rest was 18.3 months. In the *fair* category, in which the information was available in 48 patients, the average length of bed-rest therapy was 20.3 months. In this group, which comprised 80 per cent of the total cases, the over-all average time in bed was 19.5 months. In the 15 *poor* cases, the average length of bed rest was 15.4 months In the 4 *bad* cases, the average length of bed rest was 12 months. In this unsatisfactory group, which represented 20 per cent of our total number, the average period spent in bed was 14.6 months In order to test the role of bed-rest therapy in a little more detail, Figure 10 was prepared. Of the 33 cases, where 2 or more years were spent on bed-rest therapy, there were 7 *good* and 21 *fair* cases, representing 29 per cent of the total number in the satisfactory category. In the 17 patients spending from 1½ years to 2 years in bed, 15 per cent of the total number were in the satisfactory range, and the 21 cases spending from 1 year to 1½ years in bed, similarly approximately 17 per cent of the total number were satisfactory. Finally, in the 25 cases where 1 year or less was spent in bed, again about 18 per cent of the total number of cases were satisfactory. This analysis leaves no doubt that in the group in which 2 or more years of bed-rest therapy was employed, the largest number of acceptable end-results at the termination of the treatment took place

DISCUSSION

This study points out the complexities of the problem in Legg-Calvé-Perthes disease. Were a specific cause-and-effect relationship

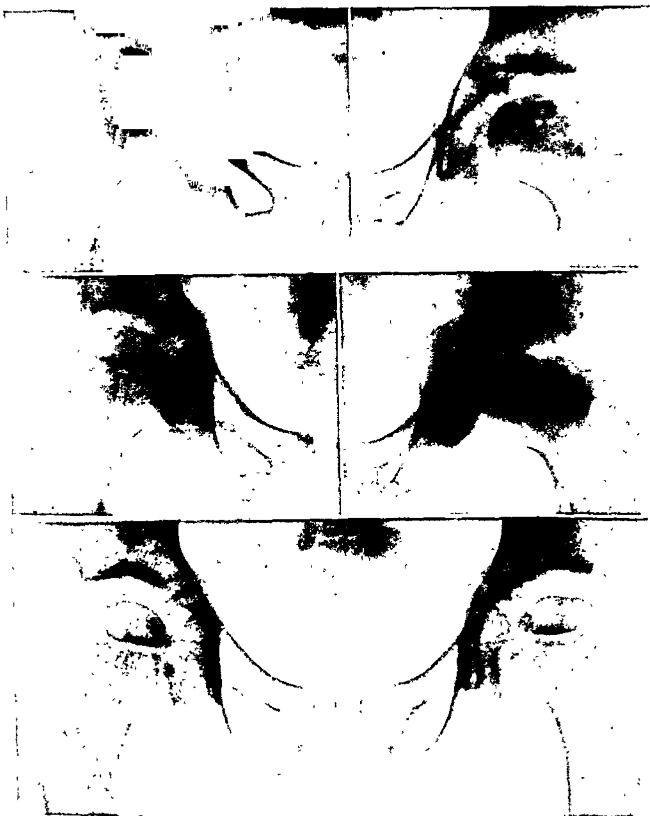


FIG. 11. D. M. This patient had had some vague symptoms referable to his left hip at 4 years of age. Roentgenograms taken on May 18, 1946, were diagnosed as early Perthes with changes of questionable degree. The patient was not treated, and presumably he remained symptom free until he jumped off a wall in October, 1949, and developed pain and limp on the left side. Roentgenograms then indicated a far-advanced Perthes' disease, for the treatment of which he was transferred to Blythedale in December, 1949. He was kept in bed until September, 1950, and then allowed up on crutches for several additional months.

He had ¾-inch shortening and 20° restriction in internal rotation with 1½-inch atrophy of the left thigh.

Epiphyseal quotient—28; this indicated a bad result and a poor prognosis for future function.



FIG. 12. A. L. Age at onset 6 years with symptoms dating back 5 weeks consisting of pain and limp on left side. The patient was admitted initially to hospital in January, 1952, and placed on bed rest and traction. This was continued at Blythedale, where he was transferred July, 1953, and remained until his discharge in March, 1955. He continued on crutches for an additional year.

In November, 1956, he was found to walk well and presented ½-inch shortening with moderate residual atrophy of the left thigh and calf. Motions were satisfactory with 10° loss of internal rotation and abduction.

The earliest roentgenograms showed a prominent metaphyseal rarefaction. Subsequently, the neck broadened and the head flattened, even though treatment was prompt. Epiphyseal quotient—62.

operating, the conclusions would necessarily be in one direction without leaving room for argument. Regrettably it must be conceded that there is considerable latitude for discussion in considering the role of therapy. In scrutinizing minutely the figures depicting the several factors affecting the prognosis, it is noteworthy how broad and extensive are the ranges and latitude. We are in complete agreement with those who hold that there is wide variability in this disease and with Goff who proposes the appellation *syndrome* rather than disease. As examples of inconsistencies, we have had patients with symptoms dating back short intervals who exhibited advanced changes in the femoral

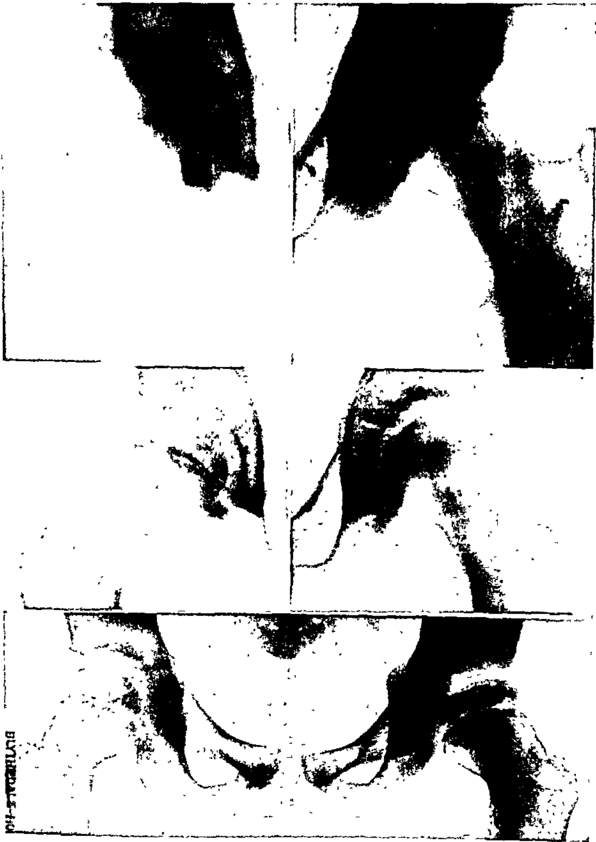


FIG. 13 J. C. Age at onset 4½ years with history of left limp of 6 months' duration. The patient was admitted to hospital August, 1941, and treated with bed rest until September, 1942. Then he was transferred to Blythedale, where bed rest was continued until February, 1943, when he was discharged.

On follow-up examination in October, 1956, he was found to be symptom free. There was ½-inch shortening of the left leg with good musculature. There was 5° loss in internal rotation. Roentgenograms showed very satisfactory result without deterioration. Epiphyseal quotient—79.

capital epiphysis (Fig. 11). We have had our share of patients who were received for therapy early in their disease, treated with bed-rest therapy promptly and consistently until termination of the active process in the femoral head and ended with a *fair* result and, at times, even a *poor* one (Fig. 12).

It should again be emphasized that this is a short-term evaluation recording the state of the femoral capital epiphysis at the termination of treatment. In the few cases that have presented the opportunity for long-term follow-up, we have been encouraged to find that early estimates of the epiphyseal quotient held up very satisfactorily in final evaluation. Instead of finding that cases deteriorated with the passing of time, surprisingly we found in many instances an improvement in the final picture (Fig. 13).

This experience coincides with that of Eyre-Brook, who found that some change for the better can result by increase in depth of the epiphysis from natural growth, especially in the younger-age group, where additional growth has yet to ensue. Contrariwise, where the immediate result was *poor* or *bad*, the follow-up result tended to be equally *poor* or *bad* (Fig. 14), sometimes worsening because of shortening of the neck. In none of the *good* and the *fair* groups was any appreciable neck shortening found to be present because of severe epiphyseal or metaphyseal change (Fig. 15).

We have purposely eliminated discussion of the changes in the cycle in the Perthes process, since these have been described very adequately and amply many times in the literature. In the cases in which the epiphysis disappeared entirely and became involved in massive thinning and waferlike fragmentation (Fig. 16), the end-result usually was poorer than in those in which the necrosis appeared to be less severe. The extensiveness of the metaphyseal rarefaction and cyst formation also provided poor prognostic signs (Fig. 16).



Fig. 14. Age at onset 9 years. Admitted to Blythedale in November, 1935. The patient was kept on bed rest until March, 1936, and in a wheel chair and on crutches until March, 1937. He was discharged from Blythedale on March 7, 1937, on full ambulation.

On last follow-up—on January 6, 1951—when he was 25 years of age, he complained of pain in the right hip on unusual exertion and in inclement weather. He had ¾-inch shortening and about 50 per cent reduction in motion of the right hip. At 18 years of age he spent a short period in the Marine Corps but received a disability discharge. The final result is poor and was forecast in the marked deformity evident in 1936. Epiphyseal quotient—33.

Our patients demonstrated variability in the length of time necessary for the cycle to be complete. In the *good* and the *fair* groups, generally the cycle ended earlier than in the *poor* and the *bad* groups. It should be recalled that in this treated group, in about half the cases the total treatment was subdivided into several categories of non-weight-bearing. In more recent years the treatment tended to more consistent total bed rest. The real purpose of this chapter rests in its prospective usefulness as a basis for comparison with large groups treated in other or similar fashion. Mindell and Sherman were quite confident that, although treatment was felt to be important, it did

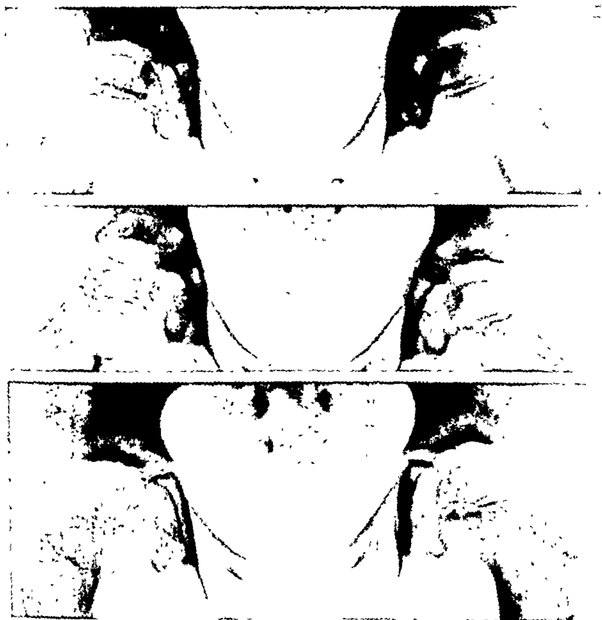


FIG 15. M. M Age at onset 5 years with history of right-sided limp not related to injury in September, 1952. Interval between symptoms and diagnosis, 2 months. Admitted to Blythedale in December, 1952, and treated with bed rest and traction until April, 1954. After an additional period of 4 months in wheel chair he was permitted free ambulation on August 8, 1954.

Early roentgenograms show minimal deformity of the right femoral epiphysis without much metaphyseal change. The roentgenogram on November 10, 1956, shows very satisfactory right constitution with mild broadening of the femoral head and neck. Epiphyseal quotient—75.

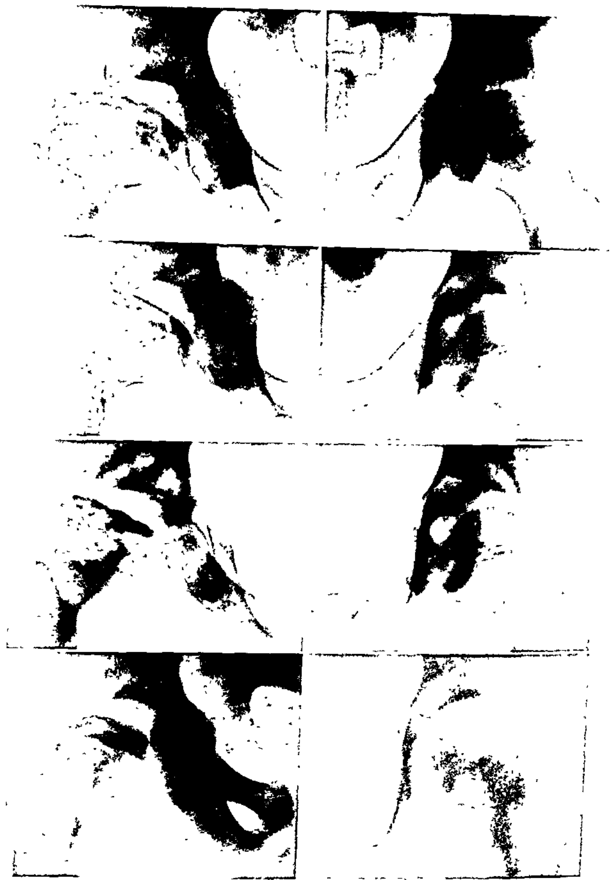


FIG. 16 P. V Age at onset 7 years and 9 months with pain and limp on the right in January, 1954 Admitted to Blythedale in December, 1954, and kept on bed rest and traction until September, 1956. At termination of treatment he presented ½-inch shortening of the right leg, 1-inch atrophy of the right thigh and 1¼-inch atrophy of the right calf. He lacked 30° in internal rotation and 15° in abduction of the right hip.
The initial roentgenograms showed a dense femoral epiphysis with concomitant meta-

(Continued on facing page)

not matter, according to their analysis, whether it was ambulatory or nonambulatory.

CONCLUSION

A statistical analysis of 100 cases of Legg-Calvé-Perthes disease has been presented. The results were obtained from roentgenograms taken at the completion of the Perthes' cycle. The method of measurement chosen was the determination of the epiphyseal quotient. This quotient stated mathematically the percentage restoration of the involved side in comparison with the normal hip. There were 11 bilateral cases with gross involvement of both hips; these were excluded from the detailed calculations made in this study.

The patients treated more recently were confined to bed with light bilateral traction. The patients treated in earlier years received some bed-rest therapy, but this was alternated with such compromise methods as the use of crutches, braces and wheel chair to avoid weight-bearing. In the total picture presented by the patients in this study, including all forms of therapy, the results obtained from roentgenographic analysis were: satisfactory, 81 per cent; unsatisfactory, 19 per cent. In the group spending 2 or more years on bed rest and mild traction, the results were satisfactory in 29 per cent. In the groups spending 1½ years, 1 year and 6 months in bed with traction, the percentage of satisfactory results was significantly lower for each subgroup. Although Legg-Calvé-Perthes disease has many variables and unpredictable attributes, amongst which may be mentioned age at onset, time when treatment was started, and severity of disease, it can be concluded that a small but definite advantage in therapy is afforded by prolonged bed rest.

REFERENCES

1. Bozsan, E. J.: A new treatment of intracapsular fractures of the neck of the femur and Calvé-Legg-Perthes disease, J. Bone & Joint Surg. 14:884-887, 1932.
2. Calvé, Jacques: Sur une forme particulière de pseudo-coxalgie greffée sur des déformations caractéristiques de l'extrémité supérieure du fémur, Rev. chir. 42:54-84, 1910.
3. Eyre-Brook, A. L.: Osteochondritis deformans coxae juvenilis or Perthes disease; the results of treatment by traction in recumbency, Brit. J. Surg. 24:166-182, 1936.
4. Goff, Charles Weer: Legg-Calvé-Perthes Syndrome and Related Osteochondroses of Youth, Springfield, Ill., Thomas, 1954.
5. Hauge, M. Foss: The treatment of coxa plana: a follow-up examination, Acta orthop. scandinav. 26:53-65, 1956.
6. Haythorn, Samuel R.: Pathological changes found in material removed at operation in Legg-Calvé-Perthes disease, J. Bone & Joint Surg. 31-A:599-611, 1949.
7. Heyman, C. H., and Herndon, C. H.: Legg-Perthes disease, J. Bone & Joint Surg. 32-A: 767-778, 1950.
8. Howorth, M. Beckett: Coxa plana, J. Bone & Joint Surg. 30-A:601-620, 1948.
9. Jonsater, Stig: Coxa plana; a histo-pathologic and arthrografic study, Acta orthop. scandinav. 12(Supp.):1-98, 1953.
10. Katz, Jacob F.: Protein-bound iodine in Legg-Calvé-Perthes disease, J. Bone & Joint Surg. 37-A:842-846, 1955.
11. Legg, Arthur T.: An obscure affection of

FIG. 16 *(Continued from facing page)*

physeal rarefaction. Periosteal duplication was already visible along the inferior margin of the femoral neck, though the head had not broadened visibly. By December 14, 1954, when treatment was commenced, severe metaphyseal rarefaction and disorganization were seen, along with marked thinning, fragmentation and lateral spread of the epiphysis. The periosteal duplication had increased many fold

At completion of therapy, the spread of the head laterally had occurred to such a degree that it formed an extra layer down the superior border of the neck and over the trochanter. *It is entirely irreconcilable to blame mechanical compression of the head against the acetabulum for this distant alteration and ectopic bone production.* Epiphyseal quotient—33.

the hip joint, Boston M. & Surg. J. 162: 202-204, 1910. (Read at the Annual Meeting of the American Orthopaedic Association, Hartford, Conn., June, 1909)
12. ———: The end results of coxa plana, J. Bone & Joint Surg. 9:26-36, 1927.
13. Lippmann, Robert K.: The pathogenesis of Legg-Calvé-Perthes disease based upon the pathologic findings in a case, Am. J. Surg. 6:785-791, 1929.
14. Mindell, E. R., and Sherman, M. S.: Late results in Legg-Perthes disease, J. Bone & Joint Surg. 33-A:1-23, 1951.
15. Perthes, G.: Über Arthritis Deformans Juvenilis, Deutsche Ztschr. Chir. 107:111-159, 1910.
16. Ponseti, Ignacio V.: Legg-Perthes Disease; observation on pathological changes in two cases, J. Bone & Joint Surg. 38-A:739-750, 1956.
17. Sjovall, Helge: Sur Frage der Behandlung der Coxa Plana, Acta orthop. scandinav. 13:324-353, 1942.
18. Sundt, Halfdan: Further investigations respecting *malum coxae Calvé-Legg-Perthes* with special regard to the prognosis and treatment, Acta chir. scandinav. 148 (Supp.):1-101, 1949.
19. Zemanksy, A. P., Jr., and Lippmann, Robert K.: The importance of the vessels in the round ligament to the head of the femur during the period of growth and their possible relationship to Perthes disease, Surg., Gynec. & Obst. 48:461-469, 1929.

Morbo Legg-Calvé-Perthes—Resultatos de Tractamento

Summario in Interlingua

Es presentate un analyse statistic de 100 casos de morbo Legg-Calvé-Perthes. Le resultatos esseva determinate super le base de roentgenogrammas prendite al completion del cyclo de Perthes. Le methodo de mesuration seligite esseva le determination del quotiente epiphysee. Iste quotiente provideva un expression mathematic del procentage de restauration del latere implicate in comparation con le coxa normal. Esseva in le serie 11 casos bilateral con grossier affectiones de ambe coxas. Istos esseva excludite ab le calculationes detaliate del presente studio.

Le patientes le plus recentement tractate esseva allectate con leve traction bilateral. Le patientes tractate in previe annos recipeva un certe therapia a reposo in lecto, sed isto esseva alternate con methodos compromissori, per exemplo, le empleo de crucias, apparatos de supporto, e chaises rolante, pro evitar le portar de peso. Le analyse del roentgenogrammas de omne le patientes de iste studio, sin reguardo al methodo therapeutic usate, monstrava resultatos satisfacente in 81 pro cento del casos e resultatos nonsatisfacente in 19 pro cento. In le gruppo tractate con reposo in lecto con leve traction durante duo annos o plus 29 pro cento habeva resultatos satisfacente. Le procentage de resultatos satisfacente esseva significativemente (e progressivemente) plus basse in le gruppos a 1½ annos, 1 anno, e ½ anno de reposo in lecto con traction. Ben que le prognose del morbo Legg-Calvé-Perthes ha multe variabiles e attributos impredicibile—etate al inception del morbo, tempore del initiation del therapia, severitate del morbo, etc.—nos conclude que prolongate reposo in lecto offere un parve sed definitive avantage therapeutic.

7

Revascularization of the Neck of the Femur in Legg-Calvé-Perthes Syndrome*

A New Surgical Technic—Experience of 80 Cases

FLAVIO PIRES DE CAMARGO, M.D.

Among all osteochondroses, undoubtedly the Legg-Calvé-Perthes disease is the one which is of most interest and has been studied more particularly. This is because, in spite of all efforts, its etiology still remains obscure. The large number of methods of treatment, conservative or surgical, have had little influence on the slow evolution of the disease, and as a result patients discontinue treatment. That is the reason for such a high percentage of bad final results with the appearance of malum coxae juvenilis when the child completes his growth.

We have been studying this disease from several angles. In 15 years we gathered about 300 patients from all strata of society and in an age range of 3 to 15 years. Many of them were followed for more than 10 years, until their growth was completed. In the great majority of cases conservative treatment was chosen, that is, rest in bed, plaster immobilization or supportive walking. Conservative therapy will give good final results when the disease is treated in the early stages, so long as there is complete joint rest and the rest has been long enough

The main clinical symptoms, pain and limping, disappear after 4 or 5 months' rest of the hip joint. In spite of this clinical improvement, the lesions, especially those of the epiphyses, continue their evolution, in three typical steps—the initial period, fragmentation, reparation. These three phases are observed by roentgenologic or pathologic examination. Generally, each stage lasts from 1 to 4 years, even 5 years in some cases.

As this evolution is slow and the clinical symptoms disappear after a few months of therapy, the majority of the patients abandon treatment. Afterward, osteoarthrosis develops slowly, with an evident clinical picture after the age of 18. All those familiar with the treatment of Perthes' disease, especially in Brazil, know the difficulties of keeping a child immobilized or maintaining him in supportive walking for months or years. A number of factors count definitely in the interruption of treatment.

Nearly all the surgical treatment proposed up to now for Perthes' disease, such as described by Kidner, Phemister, Waldenström, Levy and Girard, Steele, Howorth and Pitzen, have as the main goal the quick revascularization of the epiphyses. This revascularization slows down the evolution of the disease and avoids deformation of the epiphyses so that osteoarthrosis does not

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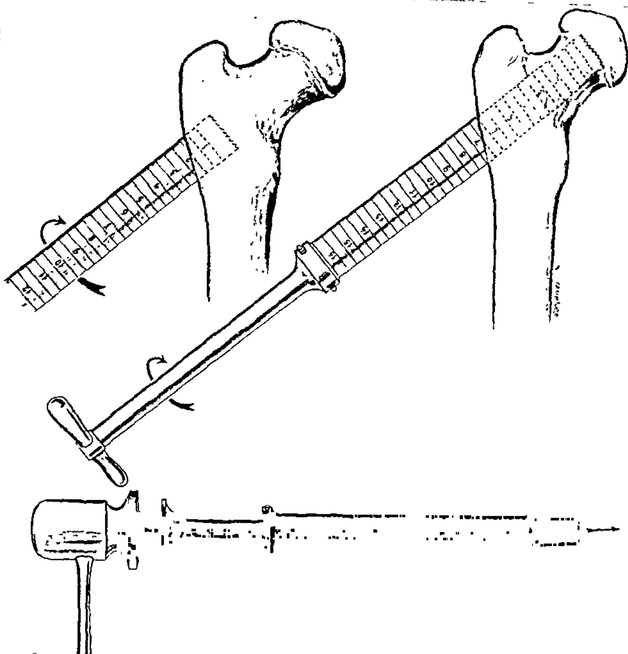


FIG. 1. *(Top, left)* The cortex of the bone is perforated, and the trephine is pushed in only a few centimeters. *(Top, right)* Once the position is correct, the trephine continues to be pushed in another 4 or 5 cm., until the epiphyseal line is reached. This is done under roentgenologic control. *(Bottom)* The bone cylinder inside then is taken out with the special impactor.

develop. All these methods have not been used routinely, due either to the insufficient number of operations performed or to the anticipated result which does not allow of our recommending them.

For the last 9 years we have used with good results a new technic which consists of re-establishing the communication between the living bone tissue of the neck of the femur and the necrotic epiphyses on the largest possible area, so as to permit a quicker revascularization. For this purpose, using a set of hollow and measured trephines, we take out nearly all cancellous bone from the neck of the femur, in thickness as well as in length, up to the epiphyseal line. The region is reconstituted, turning around the bone cylinder that was taken out and

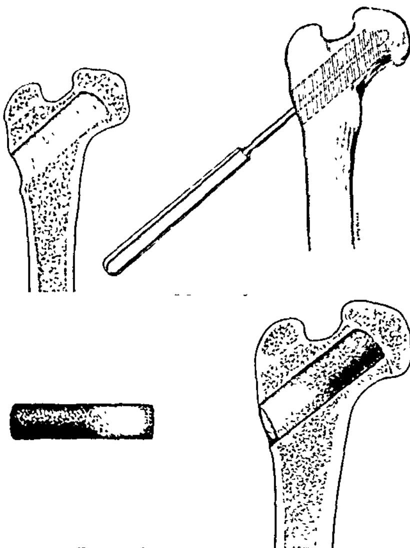


FIG. 2. *(Top, left & right)* Through the tunnel opened up in the neck of the femur, material may be taken out for pathologic examination. *(Bottom)* The bone cylinder then is put back again the other way round.

putting it back again, so that the healthy bone tissue of the trochanteric region substitutes for the diseased area of the neck.

TECHNIC

The position of the patient on the table is the same as for the operation on the neck of the femur: the inferior limbs are fixed in a moderate abduction and internal rotation. External incision is at the greater trochanter Of the trephines we choose one whose diameter is nearest that of the neck of the femur. The cortex of the bone is perforated, and the trephine is pushed in only a few centimeters (Fig 1, top, left). Roentgenologic control is one of the most important parts of the operation to ensure the correct position. Should the trephine not be in the desired position, it ought to be taken out completely and guided back again. One should not correct the position by maneuvering the trephine sideways, downward or upward. Once the position is correct, the trephine is pushed in another 4 or 5 cm. (the trephine is graduated on the outside). Another roentgenogram is taken to make sure that the trephine has reached the epiphyseal line (Fig. 1, top, right). It is then taken out with the bone cylinder inside, which is extracted with the special impactor (Fig. 1, bottom). Sometimes, when the trephine is withdrawn, the bone cylinder does not come out. In these cases the trephine is reinserted to where it was before, turning it sideways and with rotatory movements.

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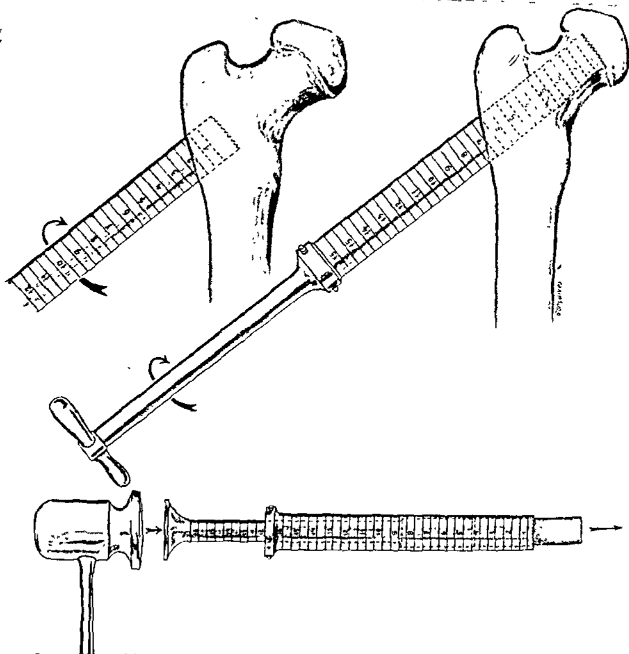


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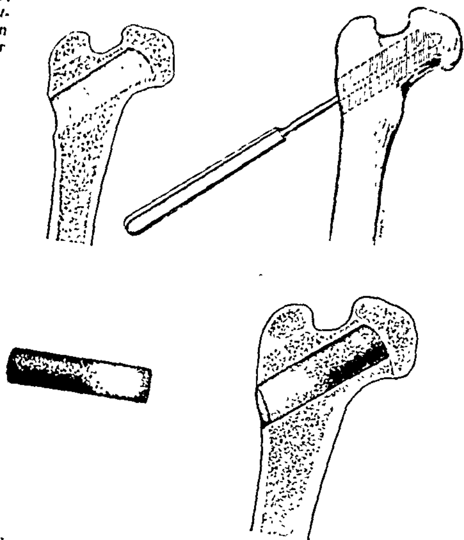


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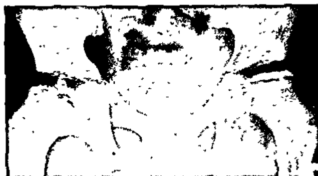
right). The bone cylinder then is put back again the other way round (Fig. 2, bottom). The operation is easy for those accustomed to surgery of the neck of the femur. The insertion of the trephine is guided by lateral and anteroposterior roentgenograms. Of course, one must be careful to avoid any



FIG. 3. Four-year-old male, April, 1953. *(Top)* Symptoms started 2 months before. Roentgenogram shows necrosis and flattening of the epiphysis on the right side. Operation performed April, 1953. *(Bottom)* Three and a half years after operation there is almost complete reconstitution of the epiphysis.









damage to the cartilage. This happens when the trephine is not well centered. In these cases a necrotic process of the epiphyses appears later and worsens the previous necrosis. This happened three times among our cases (Case No. 5).

COMMENT

Examining the patients who underwent operation we could see by roentgenogram a

FIG. 4. Nine-year-old male, January, 1950. *(Top)* Symptoms started 6 months before. Operation performed on the right side, where the lesion was worse clinically and roentgenologically. *(Bottom)* Four years after operation. In spite of the great initial flattening of the epiphysis, there was a partial reconstitution of its shape. On the left side the deformity is worse, although initially the lesion was less marked.

quick revascularization of the epiphyses, although often the deformity persisted.

How should one explain this revascularization considering that the epiphyses and the metaphyses receive their blood supply usually from widely different sources (epiphyseal and metaphyseal vessels)? This vascular system established during the growth period is not substituted, even at the adult stage, and that is why we can speak of epiphyseal and metaphyseal circulation even at an older age. Even when the epiphyseal obstacle disappears (in the bone aspect), these two sources of circulation maintain a certain autonomy. As the circulation is separated at the epiphyseal and the metaphyseal zones, we advance two hypotheses to explain this revascularization of the epiphyses with the inversion of the bone cylinder in the metaphyses:

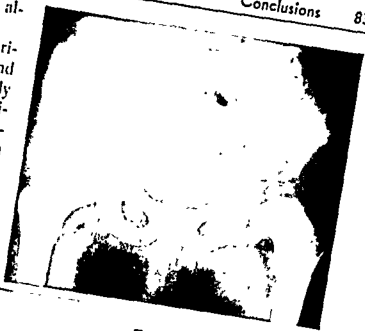




FIG. 5. Three-year-old male, October, 1953. *(Top)* Symptoms started 3 years before. Operation performed November, 1953. *(Left)* Three and a half years later reconstitution of the epiphysis is almost complete.

First, the circulation would go through the graft. Newly formed vessels would go directly, then, from the metaphyses to the epiphyses. However, this assumption does not seem to be the most acceptable, for we saw from the roentgenogram that before the graft had been completely assimilated, the epiphyses had presented evidence of revascularization.

Second, the bone graft would stimulate the formation of collateral vessels that would enter in communication with the epiphyseal circulation. This second hypothesis seems to be more acceptable. However, the circulation of this region in the child is still not very well known, there being many doubts about it. Perhaps future anatomic studies will throw light upon this subject.

CONCLUSIONS

Up to now 80 patients (table on p. 85) have been treated by this method. They ranged in age from 3 to 15. The first case was operated upon 10 years ago. The operation was performed irrespective of the different phases of the evolution of the disease, the patients remaining in plaster for 4 months afterward. At the end of this period they started to walk without support and were checked clinically and roentgenologically each 2 to 3 and 6 months until growth was completed.

Only in later years, when a good percentage of these patients complete their



permits us to draw some conclusions which we think are important and interesting:

First, in all the cases there was a quick revascularization of the epiphyses, although there was not always a reconstitution of the primitive anatomic form.

Second, in the cases in which operation was performed in the initial phases of the disease we obtained the best results. In these cases the epiphysis was not yet deformed, or at least showed little change in its form. By roentgenologic checking in

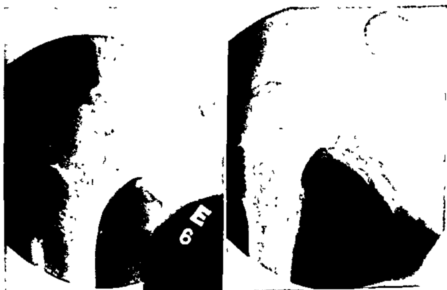


FIG. 6. Fourteen-year-old male, October, 1949. *(Top)* Symptoms started 2 years before. Roentgenogram shows serious lesion of the epiphysis of the left side. Operation performed October, 1949. *(Right)* Roentgenogram taken 8 years later. Deformity of the head of the femur, with evidence of osteoarthritis In this case the operation did not interfere with the evolution of the disease

growth, shall we be able to report results with assurance. However, what we have been able to observe in the past 9 years in the 80 patients who underwent operation





FIG. 7 Eight-year-old male, September, 1954. *(Left)* Symptoms started 2 years before. Operation performed December, 1954. *(Right)* Roentgenogram 1½ years later Partial necrosis of neck and head due to technical error. The trephine reached the articular cartilage.

Case No	Hospital No.	Age	Sex	Initial Complaint	When Symptom Was First Noted	Date of Surgery	Radiologic and Clinical Results	Comments
1	13,260	14	M	Pain	36 Months	5-13-1949	No change	
2	14,276	14	M	Pain	12 Months	10-14-1949	No change	
3	10,715	11	M	Pain	10 Months	11-11-1949	Good	
4	14,919	15	M	Pain	6 Months	11-21-1949	No change	
5	15,103	5	M	Limping	5 Months	12-16-1949	Good	
6	14,811	12	M	Limping	18 Months	12-30-1949	No change	
7	14,440	12	F	Pain	18 Months	1-13-1950	No change	
8	11,743	10	M	Limping	12 Months	1-21-1950	Good	
9	11,785	9	M	Pain	6 Months	1-27-1950	Good	Bilateral hips
10	15,569	3	M	Limping	4 Months	4-21-1950	Good	
11	13,046	10	M	Pain	12 Months	5-12-1950	Good	
12	15,898	15	M	Pain	28 Months	6-13-1950	No change	
13	16,464	12	M	Pain	12 Months	8- 4-1950	Good	
14	21,015	15	M	Pain	10 Months	8-10-1950	No change	
15	17,393	6	M	Limping	2 Months	9- 1-1950	Good	
16	16,651	11	M	Pain	18 Months	9-15-1950	No change	
17	17,910	4	M	Limping	2 Months	1-26-1951	Good	
18	18,158	14	M	Pain	12 Months	4- 6-1951	No change	
19	18,680	8	M	Limping	1 Month	6-28-1951	Good	
20	18,910	6	M	Limping	12 Months	7-13-1951	Good	
21	18,948	12	M	Pain	24 Months	7-31-1951	No change	
22	19,251	10	M	Limping	3 Months	9-14-1951	Good	
23	19,437	15	M	Pain	18 Months	10- 1-1951	No change	
24	19,337	15	M	Pain	15 Months	10-14-1951	No change	
25	20,305	9	M	Pain	6 Months	1-11-1952	Good	
26	20,133	11	M	Pain	3 Months	3-14-1952	Good	
27	20,123	7	M	Limping	24 Months	3-21-1952	No change	
28	20,266	12	M	Pain	10 Months	3-21-1952	Good	
29	21,082	9	F	Pain	4 Months	8- 1-1952	Worse	Technical error
30	21,185	10	M	Limping	8 Months	8-29-1952	Good	
31	6,459	9	F	Pain	7 Months	10- 3-1952	Good	
32	469	5	M	Limping	8 Months	11-23-1952	Good	
33	4,192	11	M	Pain	4 Months	6-12-1953	Good	
34	8,233	3	M	Limping	5 Months	10-20-1953	Good	
35	4,164	15	M	Pain	18 Months	11-10-1953	No change	
36	6,070	13	M	Pain	6 Months	12-21-1953	Good	Bilateral hips
37	4,063	9	M	Pain	8 Months	9-11-1953	Good	
38	6,047	8	M	Pain	24 Months	9-24-1953	No change	
39	6,145	5	M	Limping	24 Months	10- 1-1953	No change	
40	8,233	3	M	Limping	4 Months	10-20-1953	Good	
41	8,436	15	M	Limping	14 Months	11-21-1953	No change	Abandoned treatment
42	10,206	10	M	Limping	5 Months	12-14-1953	—	Abandoned treatment
43	12,061	7	M	Pain	12 Months	1-14-1954	—	Abandoned treatment
44	10,298	13	M	Pain	6 Months	2-12-1954	Good	
45	20,311	13	M	Limping	15 Months	4- 5-1954	No change	
46	12,357	8	M	Limping	3 Months	4-28-1954	Good	
47	2,084	8	M	Limping	8 Months	5- 7-1954	Worse	Technical error
48	12,393	7	M	Pain	1 Month	5-13-1954	Good	
49	18,028	8	F	Limping	6 Months	7- 7-1954	—	Abandoned treatment
50	20,061	8	M	Pain	12 Months	9-20-1954	—	Abandoned treatment
51	20,122	12	M	Pain	12 Months	10- 7-1954	Good	
52	20,144	9	M	Pain	7 Months	10- 9-1954	Good	
53	20,441	6	M	Pain	7 Months	10-28-1954	Worse	Technical error
54	20,301	9	M	Limping	12 Months	10-29-1954	Good	
55	20,308	11	M	Pain	4 Months	11-18-1954	—	Abandoned treatment
56	20,319	6	M	Limping	10 Months	11-19-1954	Good	
57	22,285	6	M	Limping	2 Months	12-10-1954	—	Abandoned treatment
58	20,001	8	M	Pain	24 Months	12-20-1954	—	Abandoned treatment
59	22,532	8	M	Pain	3 Months	1-17-1955	—	Abandoned treatment
60	22,583	9	M	Limping	15 Months	1-18-1955	No change	
61	22,334	3	M	Pain	4 Months	2- 2-1955	Good	
62	20,623	7	F	Limping	8 Months	2-15-1955	Good	
63	22,616	12	M	Pain	6 Months	3-16-1955	Good	
64	22,553	12	M	Limping	5 Months	5- 2-1955	Good	
65	22,690	7	M	Pain	18 Months	5- 5-1955	No change	
66	22,718	7	M	Pain	7 Months	5-18-1955	Good	
67	29,251	8	M	Pain	8 Months	6- 6-1955	Good	Bilateral hips
68	22,863	9	M	Pain	8 Months	7- 7-1955	Good	
69	28,051	7	M	Limping	7 Months	10-17-1955	Good	
70	28,252	7	M	Pain	10 Months	11-17-1955	Good	
71	28,387	14	M	Pain	5 Months	12-22-1955	No change	
72	28,408	9	M	Pain	1 Month	12-27-1955	Good	
73	28,403	12	M	Pain	1 Month	12-27-1955	Good	
74	28,420	8	F	Pain	4 Months	1- 2-1956	Good	
75	28,861	12	M	Limping	12 Months	3- 1-1956	Good	
76	28,504	6	M	Limping	2 Months	3-23-1956	Good	
77	28,896	5	M	Pain	3 Months	4- 5-1956	Good	
78	28,762	12	M	Pain	3 Months	4-18-1956	Good	
79	31,061	6	M	Limping	9 Months	6- 6-1956	Good	
80	31,871	4	M	Limping	4 Months	6- 9-1956	Good	

Summary—Total cases: 80; Male 74; Female 6; Age Group, 3 to 15 years
Results—Good: 47; No change: 21; Worse. 3, Patients lost to follow-up: 9.

series we observed its revascularization in a few months, its anatomic form being maintained.

Third, when the operation was performed in the fragmentation period or at the final stage of the disease, the results were not good. In spite of the quick revascularization of the epiphysis, it continued to be deformed, evoluting up to the final phases of growth with a typical roentgenologic aspect of an osteoarthrosis. In some cases the epiphyseal line disappeared early. Up to a certain point this is good, as it avoids a worsening of the shape of the epiphysis.

Fourth, in all cases there was an efficient clinical improvement, with disappearance of pain, limping and the contraction of the adductor muscles. This clinical improvement did not correspond to the roentgenologic aspect, where we could observe necrotic areas and large deformation of the epiphyses.

These are the principal conclusions that we have drawn to date from the cases that underwent surgery. As we have already said, only in later years shall we reach final conclusions when all the patients have completed their growth. At present, with these results, we operate only in the initial stage when the epiphysis is not very deformed, as there is no reconstitution of the anatomic form from the operation. We have also decided to lengthen the period of articular rest after the operation, which was 4 months, to 10 months. With this we hope to better the results obtained.

BIBLIOGRAPHY

Howorth, M. B.: Coxa plana, J. Bone & Joint Surg. 30-A:601-602, 1948.

Kidner, F. C.: Causes and treatment of Perthes' disease, Am J. Orthop. Surg. 14:339-345, 1916.

Levy, L. J., and Girard, P. M.: Legg-Perthes' disease, a comparative study of various methods of treatment, J. Bone & Joint Surg. 24: 663-671, 1942.

Phemister, D. B.: Operation for epiphysitis head of the femur (Perthes' disease), findings and result, Arch Surg. 2:221-230, 1921.

Pitzen, P.: Nailing to accelerate healing of aseptic bone necrosis in coxal end of femur (in coxa vara and Perthes' disease), Ztschr. Orthop. 81:7, 1951.

Steele, P. B.: Further report on the operative treatment of Perthes' disease *in* Lectures on Peace and War Orthopedic Surgery, pp. 136-143, Ann Arbor, Mich., Edwards Bros., 1943.

Waldenström, H.: The first stages of coxa plana, J. Bone & Joint Surg. 20:559-566, 1938.

Revascularisation del Cervice Femoral in le Syndrome de Legg-Calvé-Perthes. Un Nove Technica Chirurgic. Experientias in 80 Casos.

Summario in Interlingua

Depost 9 annos nos usa un nove technica pro le revascularisation del cervice femoral in le syndrome de Legg-Calvé-Perthes. Usque nunc le technica ha essite usate in 80 casos. Illo consiste del sequente manovra. Per medio de un trephina cave un cylindro es excavate ex le cervice del femore usque al profunditate del linea epiphysal. Iste cylindro comprende quasi omne le osso cancellose in le cervice del femore. Illo es revertite e re-inserite. Le resultato es que le tessuto ossee vascular in le vicinitate del linea epiphysal es remplaciate per osso salubre.

Super le base de plure annos de, rientia nos formula le sequente concl

1. In omne nostre casos un rapide revascularisation del epiphyse esseva constatate.

2. Le melior resultatos esseva obtenite in le casos in que le operation esseva executate durante le phase initial del morbo, quando le epiphyse esseva non ancora deformate.

3. Quando le operation esseva executate durante le periodo de fragmentation o le periodo final del morbo, le resultatos non esseva bon.

Iste conclusiones es non ancora definite. In le curso del niente, quando un alte procentage tes operate completa lor cresc a esser possibile rmular concl ite.

8

The Normal and the Abnormal Calcaneal Apophysis and Tarsal Navicular*

ALBERT B. FERGUSON, JR., M.D., and RALPH MAX GINGRICH, M.D.

The diseases of the tarsal navicular termed *Köhler's disease*, or osteochondritis, and *apophysitis* of the os calcis are brought together here because both should be re-evaluated in the light of present-day knowledge. Many children are subjected to unnecessary treatment because of the finding of normal changes in the tarsal navicular and calcaneal apophysis which have been confused with disease. These findings are irregular ossification of the tarsal navicular, which occurs normally in one third of the children observed, and increased density of the calcaneal apophysis in relation to the body of the os calcis, which is a normal stage of development.

Involvement of the tarsal navicular with true clinical and roentgen findings related to Köhler's disease has been seen only five times in a large pediatric orthopaedic clinic (8,624 cases per year) in 3 years. Even among those specifically presenting for foot complaints this is an incidence of only .002 per cent. Calcaneal apophysitis has not been seen at all. The cause of painful heels in children appears to lie principally in another entity described in this chapter.

THE NATURAL HISTORY OF THE APOPHYSIS OF THE OS CALCIS

The roentgenograms of the feet in the course of their development in 100 children, making up a random sample of the population of an area, have been studied in the past. From observation of these roentgenograms some statements regarding the normal development of the calcaneal apophysis can be made.

In the male the center appears between 6 and 10 years, averaging 7.9 years. The apophysis in the female was 2.3 years earlier in its appearance, averaging 5.6 years. In this series it varied between 3.0 and 8.5 years.

It takes an average of 7.1 years for the apophysis to develop and fuse to the body of the os calcis.

The growth center first appears as a few flecks of calcification of lesser density than the surrounding bone. It is located uniformly in the distal third of the posterior surface of the os calcis. The onset of this calcification usually is heralded approximately 2 to 4 years previously by the gradual development of a deep scalloping of the posterior surface of the os calcis.

For from 2 to 3 years following its first appearance the apophysis may remain in a flecky, hazy state, during which time it is not increased in density. Then it passes to a stage wherein it occupies more than one half of the posterior surface of the calcis, is increased in density over the surrounding bone, and has definite osseous texture. The bone stria of the apophysis run in its long axis.

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FIG. 1. Increased density of the developing calcaneal apophysis. This is a normal feature in the growth of this bone. *(Top)* Female, age 8. *(Center)* Male, age 10. *(Bottom)* Female, age 12.

There may be a deep and narrow cleft or clefts in the apophysis dividing it into two or three segments.

An apophysis which occupied more than one half of the posterior surface of the os calcis and yet remained in a flecky, disordered state of lesser density was not seen in this series. The apophysis was uniformly increased in density at this stage, but never so much so that bony trabeculae could not be seen.

In this homogeneous state it occupies from two thirds to three fourths of the posterior surface of the os calcis in a C shape Over the following 2 to 4 years it extends a thin finger of calcification over the posterosuperior surface of the os calcis and in 1 to 2 years thereafter fuses with it.

THE CLINICAL ENTITY

Painful Heels in Children

As every pediatrician and orthopaedist knows, painful heels in children are common. Usually this complaint is found in the over 8 year age group and centers at the posterosuperior angle of the os calcis. Two types of pathology are most usual as the cause of the complaint:

Prominent Posterosuperior Angle. The area of posterosuperior angle of the os calcis may be covered by thickened skin or callus which is tender to direct pressure and may



FIG 2. *(Left)* Prominent posterior superior angle of os calcis associated with disabling symptoms. Quotient—70 *(Right)* Postoperative view following removal of this prominent area. (Dr. William Donaldson)

appear inflamed. There is not an abnormal growth in this area, but the configuration of the os calcis and the falling away of the soft tissues about it leave this angle jutting out prominently to form a local tumor.

Prominence of the posterosuperior angle of the os calcis and secondary symptoms from irritated callus over it obviously depend on a number of factors. However, it is helpful to evaluate *the prominence of this* area of the os calcis by a ratio. This ratio is obtained by taking the length of a line (centimeters) drawn from the posterior angle of the subtalar joint line to the posteroinferior angle of the os calcis and dividing this figure into the length (in centimeters) of a perpendicular erected to the most prominent part of the posterosuperior angle of the os calcis. The quotient runs from 0.52 to 0.58 for the median group. A quotient *above* 0.59 *to* 0.60 *has been found* in those patients who have been seen because of symptoms at the posterior angle of the os calcis.

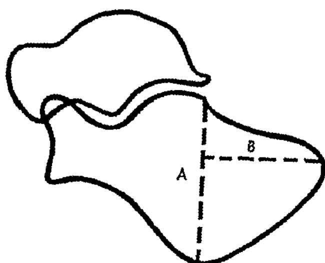


Fig 3. Method of measuring relative prominence of this area of the os calcis. A line (A) is drawn from the posterior edge of the subtalar articulation of the os calcis to the posterior inferior angle. A perpendicular line (B) is erected from this line to the apex of the posterosuperior angle. A quotient obtained by dividing *the length of (A) in centimeters into the* measurement of (B) in centimeters usually lies between 0.52 and 0.58.

If the prominence of the posterosuperior angle of the os calcis coincides with the top of the heel counter of the shoe the rubbing of this area may be enough to bring about symptoms. Such symptoms are relieved by removing the stiff counter from inside the heel leather and procuring shoes with either a higher or a lower heel upper so that pressure is not brought to bear directly on this prominence.

Achilles *Tenosynovitis.* Swelling and tenderness of the Achilles tendon immediately above its insertion are not uncommon. Symptoms in this area *apparently* are brought on by excessive activity and are relieved by rest, non-weight-bearing and heel elevation. In many cases a tight heel cord is found on physical examination limiting dorsiflexion. Such tight heel cords may be stretched once the symptoms have subsided in order to avoid a recurrence. However, heel-cord stretching when symptomatic will accentuate the symptoms.

At the time of physical examination tenderness and swelling are confined to the lower end of the Achilles tendon and appear to be consistent with a tenosynovitis. Occasionally the swelling of the distal tendon may obliterate the angle formed between it and the os calcis.



Fig. 4. Tenosynovitis of Achilles tendon at its insertion into the os calcis. Note the increased soft-tissue *swelling lying just* above the posterosuperior angle of the os calcis.

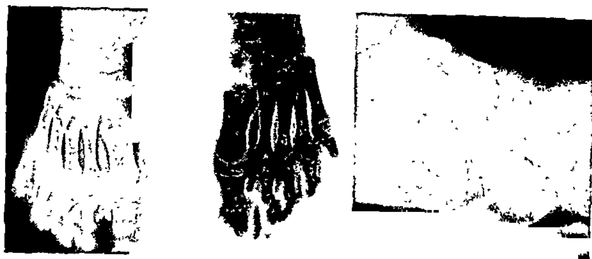


FIG. 5. The normal development of the tarsal navicular. *(Left)* Male, age 4. Irregular ossification of the navicular from multiple centers of ossification on the right. Note narrowed appearance and irregular ossification in cuneiforms. Narrowed appearance alone is not reliable. *(Right)* Female, age 2 years and 7 months. Ossification of the navicular from a single center

APOPHYSITIS

No case has been seen of swelling centering about the apophysis of the os calcis or involving symptoms related to this area. Two cases of osteomyelitis of the os calcis have been seen, but in both of these swelling and tenderness centered over the metaphyseal area of the body of the os calcis and were appreciated either laterally or medially over the os calcis. In no case has a roentgen variation from the normal heavy density been visualized from which it could be inferred that the apophysis of the os calcis was undergoing aseptic necrosis.



FIG. 6 Male, age 7 years. Density pearing in the navicular as multiple cent coalesce to form a single ossificati center.

The observation period when this entity was searched for actively involved a 3-year period and 25,874 patient observations, and during that time it was not found. As a result, it is possible to question the existence of the entity as a cause of heel pain in children.

NATURAL HISTORY OF THE NAVICULAR

In reviewing the roentgen picture of the development of 100 tarsal naviculars, the large number of instances of development of this bone by multiple ossification centers is at once apparent Thirty-seven ossified from multiple centers and a single center. equently overlap ltiple centers s a false appear eased density fragmentation. the navicular one center, of appeara s quite c

averaging 2½ years in males and 2 years 1 month in females.

The naviculars ossifying from multiple centers were delayed markedly in their appearance time by comparison. In males the average was 4 years and 4 months; in females, 3 years and 3 months. However, the multicentered type of growth rapidly became a single center at 6 years and 6 months (average) for males and 5 years for females. The time when confusion in the diagnosis of Köhler's disease might arise because of the appearance of fragmentation and increased density which multiple centers of ossification may produce is roughly between 3½ and 6½ years.

CLINICAL INVOLVEMENT OF THE TARSAL SCAPHOID

In reviewing actual cases of Köhler's disease there are features which enable this condition to be picked out from the normal variation in scaphoid ossification.

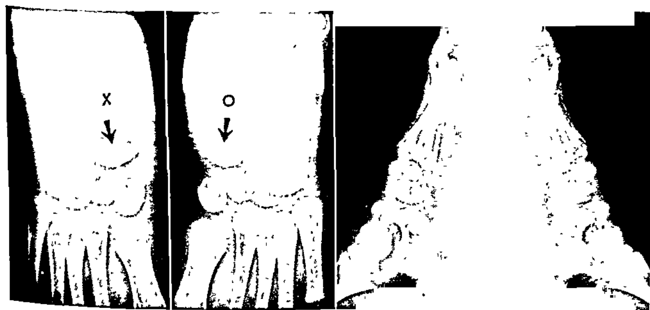
During the same period when no cases of calcaneal apophysitis were seen, five cases which were diagnosed as Köhler's disease were seen. All cases had definite clinical signs.



Fig. 7. Multiple centers coalescing, giving the navicular an irregular appearance.

The clinical signs were localized swelling and tenderness over the tarsal navicular which was most marked medially. In addition, there was peroneal muscle spasm in one foot, which limited inversion completely. Inversion often is painful, but there was no deformity of the foot associated with the disease, except in one case, with fixed eversion due to pull of the peroneals which resulted in a severely pronated foot.

The age group varied from 3 years and 6 months to 4 years and 8 months, three of



Figs 8 and 9. Kohler's disease Fig. 8 *(Above) (Left)* Anteroposterior view at onset in female aged 3½ years with soft-tissue swelling and density of the entire navicular. *(Right)* Lateral view at onset showing narrowing and increased density.

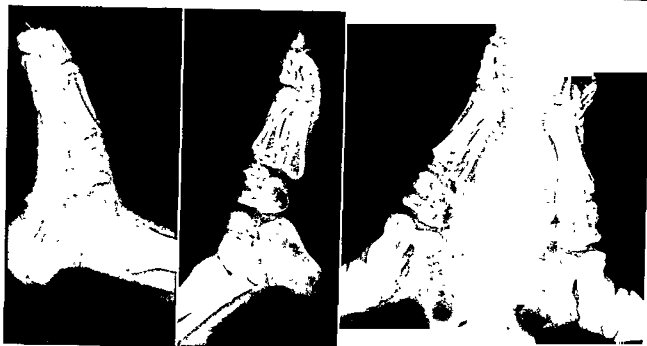


Fig. 9. *(Left)* Three months later with further narrowing and density. Note loss of convexity of cuneiform surface of navicular as compared with Figure 8 *(right)*. Other foot included for comparison. *(Right)* Six months later showing rapid reconstitution of navicular. Very minimal narrowing still remains.

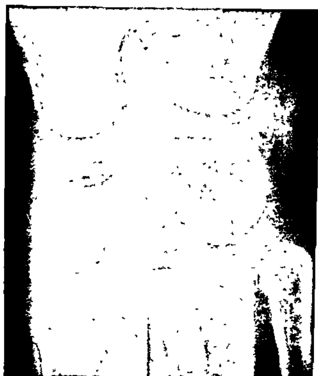


Fig. 10 Male, 4 years and 7 months. Reconstitution in Kohler's disease with initial radiolucency appearing centrally.

the four cases occurring in girls. All were unilateral.

None of the cases exhibited multicentered ossification in the opposite navicular by roentgenogram.

In all, the entire bone became dense. There was a difference in measurement of the width of the navicular running in a plane between the talus and the cuneiform of at least 2 mm. between the abnormal and the normal sides when first seen. In three of the four the difference progressed still further under roentgen observation. When the bone lost its increased density, this diameter was reconstituted rapidly to within 5/10 mm. of the normal side. All were accompanied by easily recognized deep soft-tissue swelling on the roentgenogram. Fragmentation was not a marked feature, although the increased density did not disappear throughout the bone evenly. Often radiolucency first appeared centrally.

The time interval for these changes appeared to be extraordinarily rapid when compared with conditions such as Legg-Perthes disease. One case was noted to



FIG. 11. Köhler's disease in female 4 years and 8 months of age. There was associated peroneal muscle spasm, holding foot in pronated position. *(Left)* Anteroposterior view. Note contrast in density of entire bone on left as compared with tarsal navicular on right. *(Right)* Lateral showing increased density of navicular and pronated positions of foot.

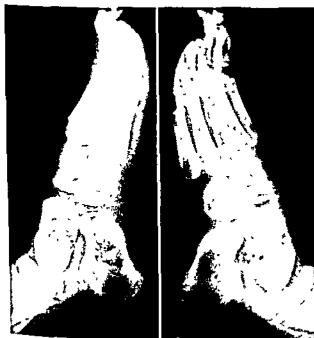


FIG. 12. Density and narrowing with soft-tissue swelling about navicular in boy aged 4 years. Involved foot, right; normal, left.

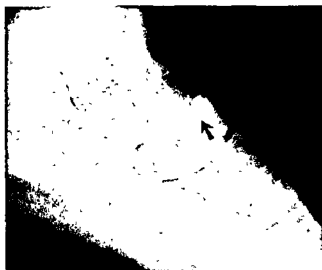


FIG. 13. Osteochondritis dissecans of the tarsal navicular with area of subchondral bone separated from parent body.

progress to still further narrowing in a 3-month period and 7 months later was reconstituted equivalent to the normal side.

Careful measurement of naviculars ossifying irregularly reveals that progressive narrowing of the bone is not a feature, although such naviculars appeared to lie on the narrow side in the lateral roentgenogram as compared with those coming in from a single center. Other diseases of the navicular, such as osteochondritis dissecans, are readily differentiated by their localized involvement on the articular surface of the navicular, the increased density of the subchondral bone tending to occur on the talar face of the navicular and to be demarcated from the parent bone by a crescent-shaped area of radiolucency.

MALES—SINGLE CENTER

AGE	LIMITS		AVERAGE
2	.1	to .3	.2
2½	.1	.5	.25
3	.1	.6	.33
3½	.1	.6	.39
4	.2	.8	.52
4½	.4	.8	.58
5	.5	.9	.64
5½	.6	.9	.7
6	.6	.9	.74
6½	.7	1.0	.78
7	.7	1.0	.84
7½	.7	1.0	.87
8	.8	1.1	.94
8½	.9	1.1	.97
9	1.0	1.2	1.04
9½	1.0	1.2	1.05
11	1.0	1.3	1.1
13	1.1	1.5	1.28

MALES—MULTIPLE CENTERS

AGE	LIMITS		AVERAGE
3	.1	to .1	.1
3½	.1	.1	.1
4	.1	.4	.27
4½	.1	.5	.32
5	.1	.65	.38
5½	.2	.7	.45
6	.2	.8	.46
6½	.3	8	.53
7	.3	.9	.58
7½	.5	.9	.69
8	.5	1.0	.74
8½	.6	1.1	.85
9	.7	1.2	.94
9½	.8	1.2	.99
11	.9	1.3	1.04
13	1.0	1.5	1.35

FEMALES—SINGLE CENTER

AGE	LIMITS		AVERAGE
1	.1	to .1	.1
1½	.1	.3	.2
2	.1	.4	.28
2½	1	.6	.36
3	.2	.6	.44
3½	.3	.7	.53
4	.3	.8	.56
4½	.4	.8	.63
5	.45	.85	.66
5½	.5	.9	.70
6	.6	1.0	.76
6½	.6	1.0	.82
7	.7	1.0	.87
7½	.7	1 1	.89
8	.8	1.1	.93
8½	8	1.15	.95
9	.9	1.2	1.04
9½	1.0	1 2	1 1
11	1.0	1.3	1.15
12	1.0	1.5	1.25

FEMALES—MULTIPLE CENTERS

AGE	LIMITS		AVERAGE
3	.1	to .4	.23
3½	.1	.6	.25
4	.1	.7	.36
4½	.1	.7	.4
5	.3	.8	.53
5½	.4	.9	.59
6	.4	.9	.63
6½	.5	1.0	.7
7	.6	1.0	.73
7½	.6	1.0	.77
8	.7	1.1	.82
8½	.8	1.1	.88
9	.8	1 2	[illegible]
9½	.8	1 2	1.02
11	1.0	1.3	1.14
13	1.0	1.5	1.3

FIG. 14 Measurements of navicular width in longitudinal plane by age, by sex and by development from multiple and single centers

SUMMARY

This chapter questions seriously the existence of such an entity as apophysitis of the os calcis. In our experience, the most common cause of painful heels in childhood is described as excessive prominence of the posterosuperior angle of the os calcis. Measures to relieve it are given. Density of the apophysis of the os calcis as an indicator of apophysitis is shown to be unreliable, since

all os calci pass through this change as part of its normal development.

Köhler's disease apparently does occur as a definite clinical and roentgen diagnosis. It should not be confused with the normal development of the os navicular from multiple centers of ossification which occurs in approximately one third of normal children. Narrowing of the navicular, increased density and soft-tissue swelling are all present with the disease. In this small series of cases Köhler's disease occurred only in naviculars developing from a single ossification center. The relatively short span of the full course of the disease is noted. The patient should be kept non-weight-bearing to allow reconstitution of the navicular. There is no clue as to the reason for this change in the navicular. Careful attention to the normal development of the navicular will prevent unnecessary treatment of the normal child.

Normalitate e Anormalitate del Apophyse Calcanee e del Osso Navicular Tarsal

Summario in Interlingua

Iste articulo questiona seriemente le existentia del entitate "apophysitis del calcaneo." Le plus commun causa de dolores del calce in juveniles es, secundo nostre experientia, un prominentia excessive del angulo posterosuperior del calcaneo. Mesuras de alleviamento es presentate. Es monstrate que densitate del apophyse calcanee es pauco secur como indication de apophysitis, proque omne calcaneos experientia iste alteration como parte de lor disveloppamento normal.

Il pare que morbo de Köhler corresponde a un definite diagnose clinic e roentgenologic. Illo non deberea esser confundite con le disveloppamento del osso navicular ab multiple centros de ossification, proque isto es occurrentia normal que pote esser observate in circa un tertio del juveniles sin pathologia. Striction del osso navicular, augmento de densitate, e tumescentia de tessuto molle es omnes presente in casos del morbo. In le presente pauco numerose serie de casos, morbo de Köhler occurreva solmente in ossos navicular que se disveloppava ab un sol centro de ossification. Le relativemente breve durantia del curso complete del morbo es notate. Le patiente debe esser protegite contra cargas de peso pro permitter le reconstitution del osso navicular. Il existe nulle signo que identificarea le rationes de iste alteration in le osso navicular. Per prestar le plus caute attention al disveloppamento normal del osso navicular on evita le tractamento innecessari de juveniles normal.

9

Avascular Necrosis of the Carpal Lunate

FREDERICK M. MAREK, M.D., F.A.C.S.*

Kienböck's disease, avascular or aseptic necrosis, osteochondritis, post-traumatic malacia of the carpal lunate, lunatomalacia, are only some of the labels applied to a lesion of the carpal lunate bone which so far has defied accurate analysis of its pathogenesis and has presented largely unsolved therapeutic problems. A voluminous partisan literature has accumulated, emanating mainly from the Scandinavian countries and Germany. There are only a few British and American reports.[5,8,14,18] I have seen seven patients with avascular necrosis of the carpal lunate in my private practice; four of these have been followed adequately and form the basis of this report.

PATHOGENESIS AND PATHOLOGY

There is now almost universal agreement that the carpal lunate infrequently becomes more or less completely necrotic. Kienböck[12] and Axhausen[1] deserve credit for establishing this firm pathologic concept. Later pathologic reports have confirmed this fact.[5,6,9,14,18,20] The traumatic etiology of this lesion is also accepted generally. Other theories, such as Axhausen's arterial embolism, infection and various constitutional abnormalities, do not seem to be tenable. However, there is very active discussion as to whether Kienbock's disease presents a simple compression fracture with nonunion and pseudarthrosis formation[2,4,7,10,11,16,17,19] or whether necrosis occurs primarily and is in turn followed by secondary compression and crumbling of the bone. In 1910 to 1911 Kienböck established the characteristics of the condition and propounded an explanation. He thought that an injury, possibly a perilunar subluxation of the wrist, interfered with the circulation of the lunate with subsequent necrosis of the bone and secondary fractures and compression. The strongest argument against this hypothesis by many authors[7,10,11,16,17,19] stems from the fact that even complete dislocation of the lunate is rarely ever followed by the classic picture of Kienbock's disease. Occasional bilater[al] cases and the frequent absence of any signi[fi]cant trauma also are cited against th[e] theory. In favor are the undeniable pr[e]ponderance of young manual laborers wi[th] the predominant right wrist involved muc[h] more frequently. Stahl[19] and Dederich[7] a[re] convinced that the entire condition is due t[o] a primary compression fracture which do[es] not heal because the wrist is not immob[i]lized. Necrosis of the bone and secondar[y] fracturing follow in due time. However, n[o] pathologic material has been presented i[n] support of this primary-fracture theory, an[d] even Stahl could demonstrate only fo[ur] plausible fresh fractures out of 184 case[s] presented. Müller,[15] Hulten[10,11] and Per[s]son[16,17] believe that a step formation at th[e] distal radio-ulnar joint with shortening o[f] the ulna as compared with the radius (th[e] so-called minus variant) plays a major rol[e] in the genesis of Kienböck's disease, wit[h] the lunate supposedly broken against th[e] protruding edge of the radius.

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Fig. 1. Enlarged views of the small periosteal volar and dorsal surfaces of the carpal lunate showing entries of vessels.

Trying to distill all these reports, I have come to the tentative conclusion that avascular necrosis occurs primarily. This may be due to solitary or repeated trauma of the bone, the vascular supply of which is as precarious as that of the proximal portion of the carponavicular, the body of the talus or the head of the femur. Examination of the lunate reveals extensive cartilaginous surfacing of the bone, which actually articulates with six other bones (Fig. 1). There is only a small dorsal and slightly larger volar surface of periosteal covering where the blood supply enters. In cadaver arterial injections, Stahl[19] in 31 experiments found only one or two narrow arteries entering the lunate from the volar side, and he was able to demonstrate only one dorsal artery. While these results are to be accepted with caution, apparently there was considerably more blood supply to the navicula in these same specimens, indicating the relative precariousness of the arterial supply to the lunate. The absence of necrosis following volar dislocations of the lunate is explained by preservation of the volar blood supply in the majority of cases. Watson-Jones[20] illustrates this with clarity and also states that in "total dislocation of the lunate" avascular necrosis is inevitable. The primary interference with the blood supply is also suggested by pathologic examination.[18] My own roentgenograms and published roentgenograms almost always suggest widespread necrosis of the lunate, quite frequently without any appreciable compression. It is also common clinical experience that, apart from an occasional chip fracture, fractures of the lunate are extremely rare. Böhler,[3] who certainly has a large fracture experience, comments that he has seen only one clear-cut fracture of the lunate (associated with a fracture of the radius.) Watson-Jones[20] does not even mention fractures of the carpal lunate in the 4th edition of *Fractures and Joint Injuries.* All seven patients seen by me recalled a definite injury to the wrist. What is even more significant is that all injuries (my case material allowed intensive questioning) consisted of forcible and excessive dorsiflexion of the wrist. It seems logical to deduce that this excessive dorsiflexion injured the important volar arteries to the lunate. Finally it is difficult to explain the clinical course of these patients on the basis of a primary fracture. Frequently the original injury is of considerable severity, but symptoms seem to diminish with and without treatment in a relatively short period of time. Many patients seem to be unaware of symptoms for months or years (unless questioned very closely) and then develop pain and disability after another minor injury, or simply after doing unaccustomed manual work. This classic clinical history and course deserve renewed emphasis, since many cases are missed and the causal relationship to the initial trauma is of legal importance. The

absence of severe pain and disability initially and in the interval stage is probably again due to the anatomy of the lunate, which has so very little periosteum (which has been demonstrated repeatedly to be the pain-sensitive structure). It is not surprising, therefore, that these frequently stoic heavy laborers do not complain severely immediately following the injury and are quite likely to forget about any such incident. In this connection I think that it is quite essential to make a diagnosis of sprain of the wrist with utmost reluctance and to roentgenograph these wrists periodically to exclude, amongst other serious injuries, avascular necrosis of the lunate. Actually there is no reason to assume that compression fractures of the lunate cannot occur, as Stahl[19] demonstrated. However, I do want to emphasize that these cases constitute a very small minority. Once the lunate is necrotic, it is quite likely that the so-called minus variant predisposes to compression of the proximal radial surface of the lunate. The position of the lunate as the "keystone" of the wrist accounts for the severe stresses imposed on the bone.

DIAGNOSIS

All my patients were quite emphatic in their complaints of pain and disability at the wrist. Symptoms were relieved temporarily by inactivity and immobilization but recurred promptly on activity. There was always some local heat, swelling, direct and indirect tenderness over the lunate and, frequently, loss of strength of the hand. Diagnosis can be suspected strongly on the basis of the dorsiflexion injury to the wrist, followed by a relatively pain-free interval or directly by persistent pain and disability of the joint Clinical findings seem to be identical in all joints. However, diagnosis rests primarily on roentgenographic examination of the wrist in multiple projections.[13] The appearance of the lunate then will vary, depending on the severity and the age of the injury. There may be early faint density of the lunate as compared with the other carpal bones, usually associated with haziness of the bone structure. Usually density is quite marked later because of atrophy of the other carpal bones. Often fissure fractures are seen only with difficulty and only on roentgenograms of good quality. Frequently fissures are transverse, but later vertical fissures and irregular oblique fissures often are noted. A mottled appearance of the bone may be due to incomplete necrosis, to compression of some bone lamellae, and in later stages to some attempts at repair, which, however, is usually most incomplete. Compression in the proximodistal axis occurs regularly, usually associated with an increase in the dorsovolar diameter. The outline of the lunate often is quite irregular with spur formation and local compression, particularly in relation to the radius. In early stages there is no significant alteration of other bones or the wrist or the forearm In late stages osteoarthritic changes are common and are of significance.

OPERATIVE FINDINGS

At operation in all my cases there were some irritative changes of the synovial lining of the wrist, frequently associated with excess joint fluid. All cultures from the joint were negative. Usually the lunate can be grossly identified without any difficulty when good exposure is obtained through an adequate mid-dorsal incision. In order to make absolutely sure about identification, it is good practice to verify the diseased bone by roentgenographic control; this certainly should obviate such difficulties as Böhler seems to have encountered. Usually the lunate is deformed, crumbly, and obviously fractured. The articular cartilages of the bone are dull, fibrillated and irregular. The necrosis is quite obvious, since no bone bleeding is encountered in the more severe and older cases. The bone can hardly ever be extirpated en masse, and usually it is necessary to remove it piecemeal. The wound is closed readily in layers. Care

should be taken to repair the cut dorsal ligament, as otherwise bowstringing of the extensor tendons will leave an unsightly, though not too important, functional defect. Pathologic examination of the removed bone showed avascular necrosis, more or less complete in all my cases. Attempt at repair was poor, usually arising from the edge of the bone by ingrowth of granulation tissue. Fractures were microscopic and macroscopic. There was never any evidence of infection or any "malacia" not explained on an avascular traumatic basis.

TREATMENT

Treatment of patients with avascular necrosis of the lunate presents great uncertainties and difficult problems. It is possible that prolonged immobilization of the wrist in an early stage of necrosis of the lunate may prevent deformity, allow improved revascularization and increase the chances of reconstitution to an essentially normal bone. Unfortunately, patients usually do not present severe symptoms early. Attention should be focused on sprains of the wrist, and early case finding should be made possible by repeated serial roentgenographic examination, even if the original roentgenographic examination of the sprained wrist were negative. It should be realized that avascular necrosis of the lunate is by no means rare. Revascularization of the lunate, unfortunately, would probably require months or years and would not be adequate in a significant percentage of patients. Treatment of the later stages of avascular necrosis of the lunate by prolonged conservative immobilization seems to me to be unsatisfactory because these deformed necrotic bones provoke reaction in the rest of the joint. For this reason also I should not favor the radial shortening advocated by Hulten[10,11] or the ulnar lengthening proposed by Persson.[10,17] These osteotomies apparently are not easy and occasionally are followed by deformity, delayed union and nonunion. When avascular necrosis is severe and accompanied by crumbling of the bone and deformity, excision of the lunate is advisable. Admittedly this is a mutilating procedure; however, it seems to give satisfactory practical results in the majority of patients. The operation is simple and should never be complicated by such catastrophes as excision of the wrong bone. Convalescence from the operation is fairly prompt, but the patient should probably not be al-



FIGS 2 and 3. Case 1. Fig. 2 *(Above)* October 2, 1941 Oblique and anteroposterior views of the right wrist showing generally increased density of lunate with haziness of bone structure. There is compression of the radial portion of the lunate with step formation. Bone fragment protrudes radially. A transverse fracture line separates the small proximal compressed segment from the main body. Two small round areas of translucency are present The distal end of the ulnar surface is 2 mm. proximal to the radius (minus variant). Proximal edge of capitate is 11 mm. from radius Width of wrist, 47 mm No arthritis.

lowed to return to heavy work for several months. Significant arthritis has not developed in my patients, nor has there been much widening or subluxation of the wrist. The capitate bone moves proximally to only a very limited extent. If arthrodesis of the wrist becomes necessary, this should not be considered a terrible tragedy. I have been impressed by the relatively small disability entailed by a wrist fused solidly in good position.

CASE REPORTS

Case 1. M.M., a male aged 21, truck driver, was first seen by me on October 2, 1941. He had fallen while playing handball in 1938. Trying to break his fall, the right wrist was dorsiflexed acutely. He had considerable pain and disability at the wrist and was treated elsewhere for an acute sprain by immobilization in a splint for 3 weeks. Then he was completely asymptomatic until he started to drive a truck and lift heavy paint supplies in May, 1941. After 2 weeks on this new job he noted increasing pain and disability at the right wrist, associated with swelling and restriction of motion.

My initial examination disclosed slight swelling of the middle of the dorsum of the right wrist. Motions of the wrist joint were severely restricted: dorsiflexion, 25° (left, 90°); palmar flexion, 5° (left, 80°); radial deviation, 5° (left, 15°); ulnar deviation, 5° (left, 15°). Motions of the right forearm were free. There was marked direct tenderness over the lunate, and by *jarring the middle finger indirect* tenderness could also be elicited.

Roentgenographic examination of the right wrist (Fig. 2) revealed almost complete necrosis of the lunate with considerable compression in the proximodistal axis and a step formation apparently due to compression of the bone by the distal end of the radius. A roughly transverse fracture line was visible. There was no evidence of any true pseudarthrosis.

A diagnosis of Kienböck's disease was made by me, and the patient's right wrist was immobilized in a below-the-elbow plaster of Paris cast for 2 months. After removal of the cast the patient was improved initially, but pain and disability of the right wrist recurred promptly. The patient then was hospitalized, and the wrist was explored through a dorsal incision on May 1, 1942. The lunate was found to be completely necrotic. There were microscopic and gross fractures with very poor repair. There was no evidence of a true pseudarthrosis * The patient had an uneventful immediate postoperative convalescence. He was inducted into the Armed Forces about 1 year after operation and was able to get through the basic training period with little difficulty. He then had a 2-year *tour of* duty in the Signal and Air Corps. Eventually he returned to his work as a truck driver and was

* Pathology of this case was described in detail by Dr F Roth [18]

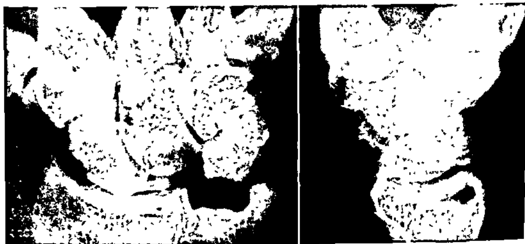


Fig 3 March 16, 1957. Fifteen years after operation. Anteroposterior and lateral views of the right wrist. Lunate is absent. There is a roughly triangular volar bone chip of fairly normal density close to radius and navicular. The capitate bone has moved to within 2 mm. of the radius. The wrist, however, has not widened appreciably; is now 50 mm. Little reactive changes at the radial surface No carpal arthritis. Minus variant has increased to 5 mm



FIGS. 4 and 5. Case 2. Fig. 4 *(Above)* April 23, 1952. Supposedly 21 days after injury. Anteroposterior, lateral and oblique views of right wrist showing generally increased density of lunate with haziness of bone structure. There is some, though slight, compression of the lunate. A roughly transverse fracture line divides the bone into a narrow, slightly ulnarward displaced proximal fragment and a triangular distal fragment. The proximal ulnar corner of the bone seems to be separated by another fissure. Minute bone chips lie between triangular and lunate and proximal to lunate. The ulna is 1 mm. shorter than the radius. Capitate is 8 mm. from the radius. Width of the wrist, 43 mm. No arthritis.

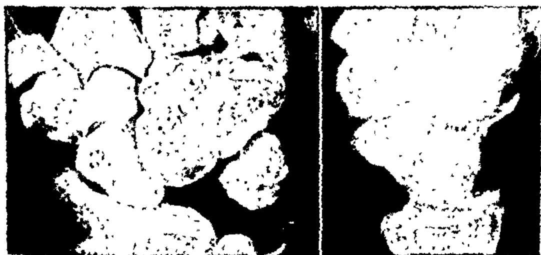
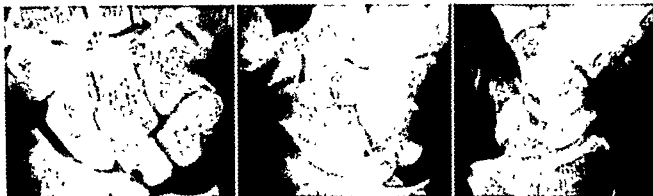


FIG. 5. April 14, 1954. Twenty-one months after operation. Anteroposterior and lateral views of the right radius Lunate is absent except for minute bone chip. Capitate bone has moved proximally 2 mm. to within 6 mm. from radius. Wrist has not widened appreciably (44 mm.). No significant arthritic changes Minus variant has increased to 2 mm

able to continue in this job requiring heavy lifting until the present. The patient thought that he favored the right wrist at times and had learned to use the left hand predominantly. He had very little pain, but occasionally the right wrist would ache on weather change

He was re-examined by me on March 16, 1957, almost 15 years after operation. There was slight atrophy of the right forearm but no appreciable swelling of the right wrist joint. Motions of the joint were somewhat restricted: dorsiflexion, 70°; palmar flexion, 25°; radial deviation, 10°; ulnar deviation, 10° Rotation of the right forearm was complete. There was no appreciable tenderness anywhere about the wrist The grip of the right hand was excellent; stronger than that of the left. Roentgenographic examination of the right wrist (Fig. 3) revealed absence of most of the lunate except for a small volar fragment. There was slight change of the relative position of the other carpal bones, but there was no evidence of any significant arthritis of the joint.

Case 2. J.P., a male, aged 25, was seen by me on April 28, 1952, for pain and disability of the right wrist. This followed forcible dorsiflexion of the right wrist when the patient pushed



FIGS. 11 and 12. Case 4. Fig. 11 *(Above)* November 6, 1954. Four months after injury. Anteroposterior and oblique views of right wrist. Lunate irregularly dense and hazy. Deep indentation of proximal surface near radius with horseshoe appearance and fairly smooth margins (other wrist showed normal lunate). Several fracture lines irregularly vertical and oblique. Cyst formation. Zero variant. Capitate 11 mm. from radius. Width of wrist, 42 mm. No arthritis.

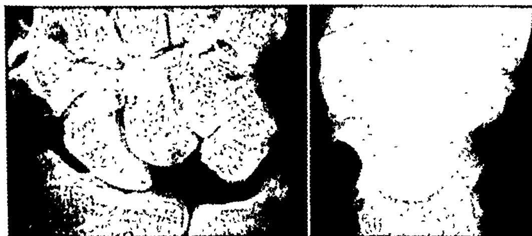


FIG 12. March 16, 1957. Twenty-eight months after excision. Anteroposterior and lateral views of right wrist. Absence of lunate. Minute bone fragments present. Capitate moved down 3 to 7 mm. from radius. Width of wrist has increased from 42 to 45 mm No arthritis

excised. The patient had a rapid convalescence and returned to light house painting about 3 months after the operation. He continued this work despite definite signs of irritation of the wrist.

He was last seen by me on March 16, 1957, when he expressed himself as being extremely pleased with the result of the operation. He had continued to work as a painter, and, because of a good deal of local construction work in his neighborhood, he frequently had to do extra heavy plastering jobs for days. Occasionally he used an elastic support but continued to use the right hand predominantly. There was very little discomfort on weather change

Examination on March 16, 1957, 28 months after operation, revealed slight soft-tissue thickening of the ulnar compartment of the right wrist. There was no definite atrophy of the forearm. Motions of the wrist were restricted: dorsiflexion, 35°; palmar flexion, 25°; radial deviation, 10°; ulnar deviation, 10° Identical motions on the left wrist. 70°; 70°; 15°; 15°. Motions of the forearm were free. Grip of the right hand was good, still considerably stronger than that of the left hand. There was no definite tenderness anywhere about the wrist. Roentgenographic examination (Fig. 12) revealed absence of the lunate with minute bone fragments in the defect. The wrist had shortened and widened slightly. There was no evidence of arthritis.

SUMMARY

The pathogenesis of avascular necrosis of the carpal lunate has been discussed and explained by traumatic interference with the arterial blood supply, followed secondarily by compression and fragmentation of the bone. The typical course of the lesion has been sketched. Treatment of later stages of avascular necrosis has been illustrated by 4 case reports.

REFERENCES

1. Axhausen, G.: Nicht Malacie sondern Nekrose des Os Lunatum Carpi, Arch. klin. Chir. 129:26, 1924.
2. Baum, E. W.: Über die traumatische Affektion des Os Lunatum und Naviculare Carpi, Beitr. klin. Chir. 87:568, 1913.
3. Böhler, L.: The Treatment of Fractures, p. 851, New York, Grune, 1956.
4. Brailsford, J. F.: The Radiology of Bones and Joints, ed. 2, p. 42, Baltimore, Wood, 1935.
5. Cave, E F.: Kienböck's disease of the lunate, J. Bone & Joint Surg. 21:858, 1939.
6. Cordes, E.: Über die Entstehung der subchondralen Osteonekrosen, Beitr. klin. Chir. 149:28, 1930.
7. Dederich, R.: Lunatummalazie — Lunatumpseudoarthrose, Zentralb. Chir. 81:248, 1936.
8. Durbin, F. C.: The early changes of Kienbock's disease of the carpal lunate bone, Proc. Roy. Soc. Med. 44:482, 1951
9. Gold, E., and Winkelbauer, A.: Mikroskopische Befunde bei Mondbeinnekrose, Arch, klin. Chir. 146:510, 1927.
10. Hulten, O.: Über anatomische Variationen der Handgelenkknochen, Acta radiol. 9: 155, 1928.
11. ———: Über die Entstehung und Behandlung der Lunatummalazie (Morbus Kienböck), Acta chir. scandinav. 76:121, 1935.
12. Kienböck, R.: Über traumatische Malacie des Mondbeins und ihre Folgezustände: Entartungsformen und Kompressionsfrakturen, Fortschr. Geb. Röntgenstrahlen 16: 78, 1910-1911.
13. Köhler, A : Borderlands of the Normal and Early Pathological in Skeletal Roentgenology, p. 88, New York, Grune, 1956.
14. Mouat, T. B., Wilkie, J., and Harding, H. E.: Isolated fracture of the carpal semilunar and Kienböck's disease, Brit. J. Surg 19:577, 1931-1932.
15. Müller, W.: Über die Erweichung und Verdichtung des Os lunatum, eine typische Erkrankung des Handgelenks, Beitr. klin Chir. 119:664, 1920.
16. Persson, M.: Pathogenese und Behandlung der Kienböckschen Lunatummalazie, Acta chir. scandinav., vol. 92, supp 98, 1945.
17. ———: Causal Treatment of Lunatomalacia, Acta chir. scandinav. 100:531, 1950
18. Roth, F. B.: Aseptic necrosis of the lunate bone, J. Bone & Joint Surg 25:683, 1943.
19. Stahl, F.: On Lunatomalacia, Acta chir. scandinav., vol. 95, supp. 126, 1947.
20. Watson-Jones, R.: Fractures and Joint Injuries, ed. 4, vol. 1, p 90, Baltimore, Williams & Wilkins, 1952.

Necrose Avascular del Osso Lunate

Summario in Interlingua

Le pathogenese e le pathologia de necrose avascular del carpal osso lunate es discutite in le forma de un revista critic del litteratura e super le base de mi restringite experientias personal.

Es concludite que vulneres per dorsiflexion del carpo obstrue le importante fluxo volar de sanguine al osso lunate. Le resultante necrose es postea sequite secundarimente, in le majoritate del patientes, per compression e fragmentation del osso. Le curso clinic es frequentemente characteristic. Le lesion initial es sequite per incerte symptomas e plus o minus non-dolorose intervallos de septimanas e mesmo annos de duration e finalmente per persistente incapacitate e dolores post un secunde lesion de grados minor o post un inusual effortio manual. Le tractamento non es multo satisfactori.

Es recommendate urgentemente que le effortio es facite de recognoscer iste condition promptemente post torquimento o luxa-

tion del carpo. In le prime stadios de necrose avascular, prolongate periodos de immobilisation debe, al minus theoricamente, haber un bon prospecto de succeder. In stadios plus avantiate, le excision del ledite osso lunate es recommendate si il existe grados significative de arthritis del carpo. Si le alterationes arthritic es multo avantiate e si le excision del osso lunate non produce un functionalmente acceptabile resultato, arthrodese del carpo es indicate.

10

Treatment of Avulsion of the Ischial Tuberosity

THOMAS A. MARTIN, M.D.,* AND GARRETT PIPKIN, M.D.†

THE PROBLEM

Opinions in the literature vary as to the necessity of treatment, other than rest, in this rare syndrome, due to failure to distinguish between the stage of apophysiolysis and that of actual avulsion.

It is our purpose here to show that when such a distinction is made, indications for treatment are clear cut and follow closely the same general principles already established for the treatment of injured traction epiphyses elsewhere in the body.

MATERIAL

The material for this chapter consists of a review of the twenty previously reported cases on which sufficient data have been recorded to permit analysis. An additional eight cases are added for study, three from the authors' private practice and five others from their private correspondence.

CLASSIFICATION

Stages of this syndrome may be divided into: (1) apophysiolysis; (2) avulsion fracture; and (3) ununited (old) avulsions (Fig. 1).

1. Apophysiolysis. Goff[6] considers the ischial apophysis an atavistic epiphysis, representing a structure that was formerly a full-fledged bone in some other species. In this particular instance he suggests a rudiment of a bone originally designed for attachment of the massive tail muscle in the larger reptilian forms.

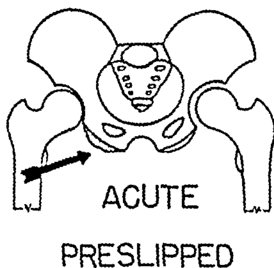
Goff continues:

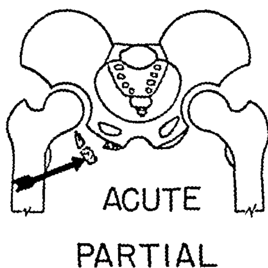
> The ossification rate of the ischial apophysis has never been timed with accuracy and, furthermore, never can be. Each individual youth will have an individual onset time, depending upon maturation rates and steroid growth factors. These disturbances are *false osteochondroses,* observed in early adolescence, and usually dependent upon trauma as an exciting agent. Such thin centers are apt to be overlooked by roentgenograms. Close studies are frequently rewarding, showing zones of irregular density, which gradually coalesce as ossification is completed Healing results when union has taken place with the main substance of the bone. Trauma is the accepted causal agent, with local tenderness and tissue thickness apparent. Sitting and forced resistance prove difficult. Usually the courses are of short duration and frequently classified as functional. The antique assumption that such a child is having "growing pains" is to disregard a possible osteochondrosis. Fortunately, bed rest and time usually bring about a complete recovery (Fig. 2 and [2,5,17,20]).

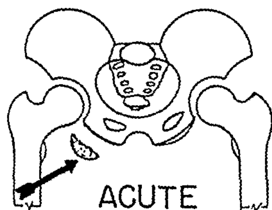
2. **Avulsion Fracture.** Such an outcome as Goff describes is not always the case. The inadequate epiphyseal plate may give way under mechanical overloading, resulting in avulsion fracture with gross separation. (See Figs. 7, left, & Fig. 8, left; Case 3). Such cases may or may not have prodromal

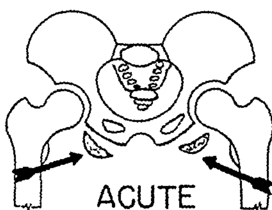
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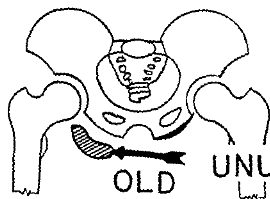
* Portland, Maine
† Kansas City, Mo











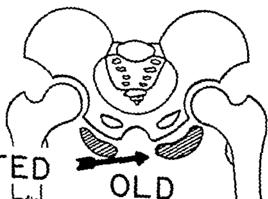


Fig. 1. Classification.

symptoms of apophysiolysis. Hellmer reported such a case with prodromal or preslipped symptoms.[10]

In his case, a 17-year-old athlete, while running the 400 meters on uneven ground, suddenly experienced a pain in his left buttock, which continued for a month. This pain was aggravated by abduction and extension of the thigh, such as used in jumping especially. Two months after the initial episode, the athlete again was running the 400 meters. After running about 40 meters, there occurred a sound like a pistol shot, which could be heard 40 meters away. The athlete experienced a severe pain in the left buttock, was unable to move and had to be transported to a hospital, where a diagnosis of avulsion of the left ischial epiphysis was confirmed by roentgenograms.

From a theoretic anatomic standpoint, an avulsion could be partial. Roughly, the ischial tuber is divided into two portions, one for the insertion of the hams, the other for the insertion of the adductor magnus.[11] Thus, a high hurdler would produce a different pattern of injury from a dancer doing a split (Fig. 2; Cases 1 & 2).

Orr[22] reports a third type of force resulting in avulsion of the ischial tubers:

> The most striking instance that I have encountered of this type were the cases of a mother *and daughter (ages 45 and 20) who were backing* their car on to a highway. It was sideswiped *from the side and rear by another car at high* speed. The women slid from the front seat, out of the side door, violently along the ground, *avulsing all four ischial tuberosities* (Fig. 3). Casts were applied, as I have previously described.[23] Good position was obtained. They made very good recoveries.

3. Old Ununited Avulsions. An untreated avulsion fracture has two possible outcomes: it may unite spontaneously, or it may go on to fibrous union with subsequent

TABLE 1. UNUNITED (OLD) AVULSIONS OF THE ISCHIAL EPIPHYSIS

AUTHOR	AGE OF ONSET (INJURY)	SEX	AGE OF PT. WHEN REPORTED	SYMPTOMS	TREATMENT	RESULT
McMaster	14	M	19	Acute lame back & hip	8 weeks' bed rest	Evacuated as unfit for combat duty
Scott	15	M	22	Pain in thoracic spine attributed to old vertebral epiphysitis, limp & 1½" shortening		Evacuated from combat area
Labuz	16	M	24	Pain on stooping & on sitting	Diathermy	Assigned to limited service
Abate	16	M	22	Pain on sitting & rising	Rest, heat, procaine	Symptoms continue
Winkler & Rapp	14	M	39	Pain on sitting	Excision of tuber	Relieved of symptoms
Berry	23	M	26	Pain & disability	Excision of tuber	Relieved of symptoms
Lewin & MacLeod	17	M	18	Pain on running or sitting	Excision advised	Not recorded
Hamsa*	Unknown	M	24	Pain on sitting	Excision advised	Unknown
Ullrich*	16	M	23	Pain on working & sitting	Excision advised	Unknown
Pipkin*	15	M	15½	Pain on sitting & working. Backache. Growth discrepancy	Excision of tuber	Relieved of symptoms

* Not previously reported

FIG. 2. A graphic record of athletic stress on the ischial tubers. (Wide World Photos, New York)



FIG 3. Avulsion fracture of all four ischial tubers in mother and daughter in automobile accident.

tuber enlargement. A summary of the "old" cases is contained in Table 1. These cases all represent avulsions of the ischial epiphyses, which developed a fibrous union with enlargement of the tuber. The symptoms of these cases are all very similar; viz., inability to sit comfortably on the enlarged, ununited tuber, and pain with lameness in back or limb, especially when under burden. The subsequent enlargement of the tuber may be so gross and irregular as to suggest a tumor. The case illustrated in Figure 4 was referred as a possible sarcoma.[23] Growth discrepancies of the limb or back may develop (Case 3 & [24]).

The "old" cases represent a *group of* fibrous unions which have a follow-up period varying from one to twenty-five years which became, or continued to be, symptomatic. Hence, fibrous union cannot be considered the criteria of a satisfactory result. Particularly noteworthy is the failure of this group of "old" cases to stand up under the stress of military duty.[1,13,16,24]

Bechtol[2] suggests even another possible disability as a result of such an injury, namely, improper prosthesis fitting, should such a case at a later date become an amputee. We quote:

> I have had a considerable interest in the ischial tuberosity, since it is the main point of weight bearing in the suction-socket type of artificial leg.
>
> One observation of some interest in the amputee cases has been that the majority of ischial tuberosities do not have muscle attachment extending to their posterior angle. This area, which is free of muscle attachments, is a very satisfactory weight bearing area. There is, however, somewhere between 10 and 20 per cent of such cases who have muscles attaching clear back on the ischial tuberosity. This muscle attachment is tender and is not satisfactory for weight bearing, and such cases require a different type of fitting in the socket.
>
> I pass this bit of ischialteric information on to you at no extra charge.

COMMENT

When the existing cases are divided into two major categories, apophysiolysis and avulsion fractures (Tables 2 & 3), the question as to whether or not more definite treatment than rest should be instituted is immediately clarified.

Cases of apophysiolysis obtain excellent results on a protective program (Table 2). Cases of avulsion fractures have all required more definitive treatment or fibrous union has resulted (Table 3). Thus, no actual discrepancy exists in the literature regarding indications for treatment.

TREATMENT

Treatment is divided into three categories, dependent upon the diagnosis, and should be governed by the same principles which have



FIG. 4 Old ununited avulsion fracture with tuber enlargement. (H. F. Ullrich)

TABLE 2. CASES OF APOPHYSIOLYSIS

AUTHOR	PATIENT'S AGE	PATIENT'S SEX	INJURY	TREATMENT	RESULT
Castellana	15	M	Jumping	Cast	Reported satisfactory
Cohen	16	M	Foot caught in a hole	3 weeks' rest	Bony union
Goff*	14	M	Football	Progressed through avascular necrosis	Bony union
Milch	12	F	Split dancing	None	Bony union
Milch	16	M	Multiple, athletic	Rest	Bony union
Hamsa*	16	M	High jumping	Rest	Presumed to be satisfactory
Urist*	Soldier	M	Not stated	6 weeks' rest	"Slight displacement—reattached"

* Not previously reported

TABLE 3. CASES OF AVULSION FRACTURE

AUTHOR	PATIENT'S AGE	PATIENT'S SEX	INJURY	TREATMENT	RESULT
Gutschank	17	M	Running (bilateral)	Unsuccessful attempt at closed reduction	Fibrous union with enlarged tubers and peri-ischial soft-tissue calcification
Hellmer	17	M	Running	Open reduction	Excellent; resumed competitve sports in 5 mos.
Karfiol	19	M	Running fast	None for 5 mos. Fibrous union found at operation. Tuber removed.	Excellent after 8 weeks. Resumed sports.
Martin*	16	M	Running	Open reduction	Excellent; 5-yr. follow-up
Milch	11	F	Dancing split	Bed rest for 4 weeks	4-week follow-up only
Mooney	16	M	Football	Plaster spica 4 weeks	Fibrous union
Mooney	16	M	Football	Taping and crutches	Fibrous union
Orr*	20	F	Auto (bilateral)	Double abduction casts with good position	"Very good recovery"
Orr*†	45	F	Auto (bilateral)	Double abduction casts with good position	"Very good recovery"
Pipkin*	14	F	Dancing split	Spontaneous anatomic reduction, plaster spica	Bony union; follow-up 10 yrs. later reports that she is a professional dancer in Paris.

* Not previously reported
† See text

been established for the treatment of injured traction epiphyses elsewhere in the body.

1. Cases of apophysiolysis uniformly do well on a rest and protective program. Ideally, reasonable bony union should be demonstrable by roentgenograms before strenuous exercise is permitted. Milch's (Figs. 5 & 6) excellent follow-ups have indicated that this may require from 2 to 4 years in the younger age groups. Urist



FIG. 5. Apophysiolysis. (Milch, Henry: Clinical Orthopaedics No. 2, Philadelphia, Lippincott)



FIG. 6 Same case as in Figure 5 two years later. Bony union of the epiphysis with slight deformity of the tuber. (Milch, Henry: Clinical Orthopaedics No. 2, Philadelphia, Lippincott)

records excellent recovery in the older group in 6 weeks.

Failure to follow a protective program could result in an avulsion fracture of the apophysis from a second injury.

The possibility of contralateral involvement should be kept in mind. Gutschank[8] recorded a case with right-sided symptoms. Roentgenograms confirmed the diagnosis and, in addition, revealed identical involvement of the left apophysis. Concurrent involvement of epiphyses elsewhere in the body has been reported by Scott.[24]

Medical management with small doses of certain antibiotics (postulated growth factor) and vitamin B_{12}, as advocated by Goff,[7] would appear to be a worth-while adjunct.

2. Cases of avulsion fracture should be treated as a fracture and reduced anatomically either by closed or open reduction. Thus far, no one has reported a purposeful closed reduction in the literature.



FIG. 7. *(Left)* Avulsion fracture of ischial tuberosity in an 11-year-old girl. *(Center)* Anatomic restitution by spontaneous reduction. *(Right)* End-result 2½ years later.



FIG. 8. *(Left)* Avulsion fracture of ischial tuberosity in a 16-year-old boy. *(Right)* Anatomic restoration by open reduction and internal fixation.

Spontaneous anatomic reduction of an avulsion fracture occurred in one of our cases.

Case I. G. S., a 14-year-old girl, doing an acrobatic split at a lawn fete, slipped on attempted recovery on wet grass, experienced a sensation of something snapping and thereafter developed a severe pain in her right buttock. She lay on the grass until transported to a hospital by ambulance, where roentgenographic examination disclosed an avulsion of the right ischial epiphysis (Fig. 7). She was administered a hypodermic of morphine sulfate, gr. 1/6, and sent to her ward on a stretcher While being assisted from the cart to a bed, she felt another sudden snap in her buttock, experienced great relief from pain, and developed ability to move her injured extremity freely. She slept well without further sedation. Fluoroscopic check the next morning, at the time she was scheduled for attempted closed reduction, showed that the avulsed epiphysis had already been reduced spontaneously. The patient was placed in a walking hip spica, extending from ankle to nipple line, which she wore for a month. A roentgenogram made 48 hours after injury, through her plaster hip spica, showed that the avulsed epiphysis had been restored to its anatomic position. After removal of her cast, she was placed on graduated exercises for 6 months.

A roentgenogram 2½ years after injury showed that the epiphysis had united solidly and without deformity (Fig. 7, *right*). She was not available for follow-up 10 years later, as she was then a professional dancer in Paris.

This fortunate case indicates that an attempt at manipulative reduction, under anesthesia, with one finger in the rectum, should be worth while.

Fresh cases of avulsion fracture which cannot be reduced by closed methods should be treated by open reduction and internal fixation. Milch,[20] although he has never reported attempting such a case, states categorically that "surgical reattachment is not to be recommended. It is technically difficult and would seem to be contraindicated." We regard this as an unfortunate statement and an unwarranted conclusion. Using Milch's approach[18,19] to the ischium, we consider open reduction of an avulsion fracture a relatively straightforward procedure for an experienced bone surgeon. The following case is illustrative (Fig. 8):

Case 2. On May 24, 1952, W. F., a 16-year-old male, while running developed a severe pain in his left hip, referred into the left buttock. He was transported by ambulance to the hospital. He was unable to move his leg, which was held in abduction, with slight flexion of the hip and the knee. There was exquisite tenderness and pain on any attempted rotation of the left hip. Locally, there were swelling, fullness and exquisite tenderness of the left buttock. Roentgenograms revealed an avulsion fracture of the left ischial tuberosity (Fig. 8, *left*). Open reduction was performed on May 29, 1952, using Milch's approach. The avulsed fragment was reattached by two stainless steel screws The immediate fixation was so satisfactory that no postoperative cast was needed. The patient was discharged ambulatorily on the tenth postoperative day and graduated to light athletics in 8 weeks. He returned to a full athletic program the next fall (8 months after operation).

The other open reductions of avulsion fractures reported in the literature have turned out equally well (Table 3).

3. Symptoms of "old" ununited cases of ischial epiphyseal avulsion are relieved by excision of the ununited tuber and repair of the tendinous origin of the hams (Table 1). The following case is also pertinent:

Case 3. D. T., a white 15-year-old male, had prodromal vague pain in the general area of his right hip for 2 months. One day, while broad-jumping, he felt a sudden snap in the right buttock and developed a severe pain in this area. He was carried to a physician, who diagnosed "severe sprain." The doctor did not take roentgenograms and gave diathermy treatments over the next 3 weeks Partial recovery gradually resulted so that the boy could walk and even run a little. He could not sit with comfort, and on moderate exertion he had pain.

Six months after injury the patient began to develop backache so that his family took him to a chiropractor. The chiropractor noted that the boy had a slight curvature and that his right leg was ½ inch longer than the left. Survey roentgenograms of spine and pelvis revealed the enlarged mass of an avulsed and ununited ischial epiphysis. Consultation with a bone surgeon was advised.

When first seen, additional medical history was of interest in that he had grown between 3 and 4 inches during the previous 6 months and had gained between 30 and 40 lbs. in weight. Physical examination revealed a well-built and well-proportioned, almost mature, white male, height 6 feet 2 inches, weight 165 lbs. He localized his pain and discomfort to the right ischial tuberosity and, more vaguely, to his low back Pressure on the right ischial area did not elicit tenderness. He volunteered that it required from 10 to 15 minutes of sitting to produce pain. There was a palpable enlargement in the right ischial area. Motion of the right hip was not limited. There was ¾ inch atrophy of the right calf and 1 inch atrophy of the right thigh The right inferior extremity was ½ inch longer than the left On standing, this increase in length produced a pelvic obliquity with subsequent compensating slight scoliosis. The scoliosis was relieved by recumbency.

Roentgenograms revealed an enlarged avulsed ischial epiphysis with peri-ischial calcification

Surgical removal of the avulsed mass, with repair of the tendinous origins, relieved the boy's symptoms.

Sufficient follow-up time has not elapsed to report on the effect of this repair on the boy's growth discrepancies.

Some patients who have symptoms from an "old," ununited avulsion are apparently unwilling to undertake the expense and haz-

ards of surgery. Presumably they have modified their activity so as to obtain comfort (Table 1).

CONCLUSION

1. Sharp distinction should be made between cases of apophysiolysis and avulsion fracture of the ischial epiphysis.

2. Cases of apophysiolysis usually progress to bony union without deformity on a rest and protective regimen.

3. A case of spontaneous reduction of an avulsion fracture of the ischial tuberosity is reported. We believe this to be the first reported case of closed reduction.

4. Accumulated experience indicates that unreduced avulsion fractures of the ischial epiphysis result in fibrous union, uncomfortable for sitting, and painful under burden. The bony fragment enlarges so that it forms an ischial mass. Subsequent growth discrepancies of the limb and the back have been reported.

5. In acute cases, should an attempt at closed reduction fail, the epiphysis should be restored anatomically by open reduction and internal fixation.

6. Symptoms in "old" ununited cases of ischial epiphyseal avulsion are relieved by excision of the ununited tuber.

REFERENCES AND REPORTED CASES

1. Abate, Charles C.: Avulsion fracture of the ischial tuberosity, J. Bone & Joint Surg 27:716-717, 1945.
2. Bechtol, Charles O.: Personal communication.
3. Berry, J. M.: Fracture of the tuberosity of the ischium due to muscular action, J.A.M.A 59:1450, 1912
4. Castellana, A.: Apophysiolysis, Rev d'orthop. 34:145-155, 1948
5. Cohen, H. H.. Avulsion-fracture of the ischial tuberosity, J. Bone & Joint Surg 19: 1138, 1937.
6. Goff, C. W.: Legg-Calvé-Perthes and Related Osteochondroses of Youth, Springfield, Ill., Thomas, 1954.
7. ——— Growth Acceleration in Legg-Calvé-Perthes Syndrome by Complementary Feedings of Aureomycin, Clinical Orthopaedics No. 6, p. 1107, Philadelphia, Lippincott, 1955.
8. Gutschank, A.: Doppelseitige Abrissfraktur des Tuber Ossis Ischii, Arch. orthop. u. Unfall-Chir. 33:256, 1933.
9. Hamsa, W. R.: Personal communication.
10. Hellmer, H.: Ein Fall von traumatischer Ablösung der Epiphyse des Os Ischii, Arch. orthop. u. Unfall-Chir. 34:45, 1933.
11. Jones, F. W.: Buchanan's Manual of Anatomy, ed. 8, p. 320, Baltimore, Williams & Wilkins, 1950.
12. Karfiol, G.: Abrissfraktur des Tuber Ischiadicum. Sportverletzung. Zentralbl. Chir. 57:2466, 1940.
13. Labuz, Eugene F.: Avulsion of the ischial tuberosity, *J. Bone & Joint Surg. 28*:388-389, 1946.
14. Lapidari, M.: Un raro caso di distracco traumatico del nucleo di ossifica zione della tuberosita ichiatica; Attic Memorie della *Soc. Lombarda di Chir.; 5 No. 17, 1937.* Quoted by Castellana.
15. MacLeod, Sidney B., and Lwein, Philip: Avulsion of the epiphysis of the tuberosity of the ischium, J.A.M.A. 92:1597, 1929.
16. McMaster, Paul E.: *Epiphysitis of the* ischial tuberosity, a case report, J. Bone & Joint Surg. 27:493-494, 1945.
17. Milch, Henry: Avulsion-fracture of the tuberosity of the ischium, J. Bone & Joint Surg. 8:832-838, 1926.
18. ———: Partial resection of the ischium, J. Bone & Joint Surg. 17:166, 1935.
19. ———. Resection of ischium *in* Operative Orthopedics, pp. 776-779, St. Louis, Mosby, 1939.
20. ———: Ischial Apophysiolysis, A New Syndrome, Clinical Orthopaedics No. 2, pp. 184-193, *Philadelphia, Lippincott,* 1953.
21. Mooney, V : Avulsion fracture of the tuberosity of the ischium, Pennsylvania M. J. 50:1072-1073, 1947.
22. Orr, H. W.: Personal communication.
23. ———: Treatment of compound fractures of the pelvis, Surg , Gynec. & Obst. 54:673-679, 1932.
24. Scott, W.: Non-union of the ischial tuberosity associated with epiphysitis vertebrae, J. Bone & Joint Surg. 28:862-864, 1946.
25. Ullrich, Henry F.: Personal communication.
26. Urist, M. R : Personal communication.
27. Winkler, Harry, and Rapp, Ira H.: Ununited *epiphysis of the ischium, J. Bone &* Joint Surg. 29:234-236, 1947.

Le Tractamento de Avulsion del Tubere Ischial

Summario in Interlingua

Pro objectivos therapeutic, apophysiolyse e fractura avulsional del epiphyse ischial es distinguite nettemente.

Casos de apophysiolyse debe esser tractate como un osteochondrosis que require reposo e un regime protective. Union ossee con deformitate minimal es usualmente le resultato.

Multiple experientias indica que nonreducite fracturas avulsional del epiphyse ischial resulta in union fibrose que causa disconforto in seder e dolor sub carga. Le fragmento ossee se aggrandi de maniera que illo forma un massa ischial. Discrepantias de crescentia secundari in extremitate o dorso pote evenir. Casos acute debe esser tractate como fracturas requirente un reduction claudite o aperte e un adequate periodo de immobilisation subsequente.

Symptomas in "ancian" casos de nonunion es alleviate per excision del non-unite tubere.

11

Epiphyseal Injuries About the Hip Joint*

W. R. HAMSA, M.D.

Considering the frequency and the varying force of muscular contraction, traumatic incidents in the muscular, the tendinous or the epiphyseal area are rare indeed. Today's athletic endeavors are commonplace, and the resultant "Charley-horse" episodes are not particularly disturbing to either patient or physician. Although one may assume that most injuries about the hip in the child or the adolescent are limited to soft tissues, with a reputation for rapid recovery, the force may expend itself in a growth plate, either epiphysis or apophysis. Early recognition of the latter group is paramount if serious disability is to be avoided. This recognition follows if the growth in pelvis and femur is visualized and related to the age of the patient under consideration.

The fetal cartilaginous pelvis and femur change into mature bone by an orderly sequence of growth in primary and in secondary centers of ossification; these centers develop in the prenatal and the postnatal periods, respectively.[1] Primary centers, present at birth, appear in the femoral body at 7 weeks, in the ilium at 9 weeks, in the ischium at 3 months and in the pubis at 5 months (Fig. 1). Subsequently three secondary centers appear in the upper femur, uniting with the body center when growth is completed. The femoral head, the greater trochanter and the lesser trochanter centers appear at 1 year, 4 years and 13 years, respectively, fusing with the body of the femur at 20, 19 and 18 years, respectively (Fig. 2). Two or three centers appear in the triradiate cartilage at 12 years, fusing ilium, ischium and pubis together at 17 years. Centers for the iliac crest, the antero-inferior iliac spine, the ischial tuberosity and the symphysis pubis appear at puberty, fusing at 20 to 25 years (Fig. 3). Prior to the time of bony union, all cartilaginous areas are vulnerable to injury.

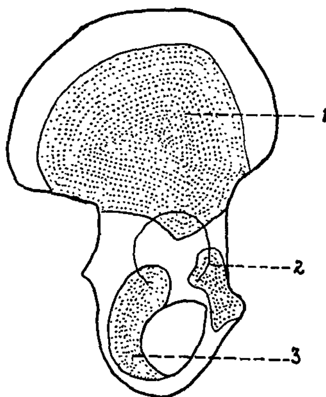


FIG. 1. Prenatal appearance of centers: (1) ilium at 9 weeks; (2) pubis at 20 weeks; (3) ischium at 12 weeks.

* From the University of Nebraska College of Medicine and The Bishop Clarkson Memorial Hospital, Omaha, Nebraska

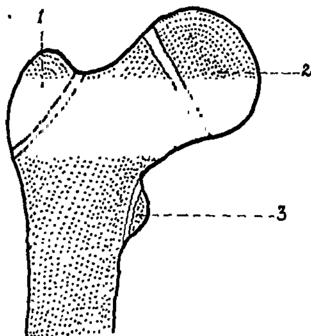


Fig. 2. Appearance and fusion ages of centers: (1) greater trochanter, 4 to 19 years; (2) head, 1 to 20 years; (3) lesser trochanter, 13 to 18 years

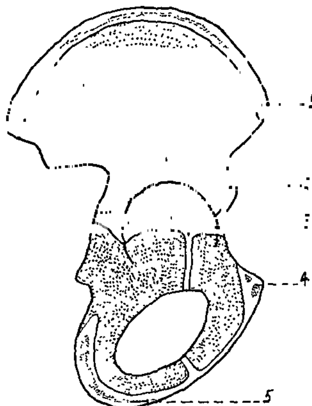


Fig. 3. Secondary centers at age 13 years: (1) anterosuperior spine; (2) antero-inferior spine; (3) triradiate cartilage; (4) *pubis*; (5) *ischium*

UPPER FEMORAL EPIPHYSIS

The gradual or the sudden slip of the upper femoral epiphysis is common between the ages of 10 and 17 years. The preponderance of males involved suggests a traumatic etiology. Endocrine deficiencies, however, may contribute to this change, as evidenced by the frequently associated obesity and hypogonadism. The traumatic change may be preceded by a period of rapid growth in an apparently normal but tall angular adolescent.[2] Here the susceptibility of the epiphyseal plate to traumatic changes is understandable. Early symptoms are pain, limp, stiffness and deformity. Associated findings of tenderness over anterior joint capsule, limited internal rotation and abduction, and muscle spasm suggest detailed roentgenographic studies of both hips in extended and in flexed positions. The latter view is particularly important in the early stages, as asymmetry of the femoral head-neck alignment and widening of the epiphyseal line are readily discernible (Fig. 4).



Fig. 4. Anteroposterior and lateral views of early slipping upper left femoral epiphysis.

Protection in this early slipping stage by crutch support or brace may suffice in co-operative youngsters. The un-co-operative and physically vigorous individual with similar early findings is a candidate for skeletal fixation by pins, screws or triflanged nail. Obvious displacement of the femoral head is treated by early manipulative reduction supplemented by open skeletal fixation (Fig. 5). Inability to reduce the displacement by closed maneuvers suggests fixation in a double hip spica cast in best obtainable position until healing occurs. Should the final healing be unsatisfactory, a late intertrochanteric osteotomy will realign the femoral neck and head functionally. Early wedge resection of the femoral neck to obtain good head and neck relationship is followed all too often by late aseptic necrosis of the femoral head.



FIG 5. Slipped upper femoral epiphysis before and after closed reduction and skeletal fixation.



Fig. 6. Healed avulsion of lesser trochanter apophysis.

LESSER TROCHANTER APOPHYSIS

This apophysis is injured rarely; the author has seen only one patient with this change. A 19-year-old male, complaining of recurring pain in his right groin of one-year's duration, dated the onset of discomfort to a violent exertion while high jumping. Recurrent attacks of pain followed each jump. The usual trainer's massage for a "Charley-horse" failed to relieve him On examination the hip joint was free. Pain was *reproduced by active hip flexion* against resistance. Roentgenographic study demonstrated a lesser trochanter elongated upward in the direction of the iliopsoas tendon with irregular outline (Fig. 6). Suspecting a healing stage of a partial avulsion of the apophysis, all physical exertion was discontinued for 4 months. Symptoms regressed, and the hip joint regained full strength without pain.

THE ANTEROSUPERIOR ILIAC SPINE APOPHYSIS

This prominence develops as a part of the iliac crest. Sudden powerful flexion of the *hip places heavy stresses at both anterosuperior* and at antero-inferior iliac spines through the tendons of rectus femoris heads and of sartorius muscle groups. The anterosuperior spine is injured much oftener. A





Fig 7 *(Left)* Acute avulsion anterosuperior iliac spine apophysis *(Right)* Healed displaced avulsion anterosuperior iliac spine apophysis.

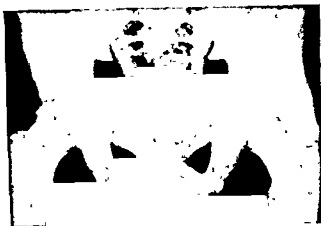


FIG. 8. Healed displaced avulsion antero-inferior iliac spine apophysis. (Milgram, J. E.: Am. Acad. Orthop. Surgeons Instructional Course Lectures 10:240)



FIG. 9. Displaced avulsion ischial tuberosity apophysis.

classic picture is the 15-year-old sprinter who felt a sudden sharp pain in the groin on leaving the blocks at the starter's gun. This pain was reproduced by active flexion of the hip and tenderness localized over a swelling above the hip joint. Roentgenographic studies demonstrated a separation of the anterosuperior spine portion of the iliac crest (Fig. 7, *left*). Following a week of bed rest with the hip in flexion, recovery occurred in 6 weeks.

Of interest is an incidental finding in an adult male of a long bony bar in the region of the anterosuperior iliac spine. This bar was asymptomatic. Its etiology was interpreted as trauma to this apophysis in puberty (Fig. 7, *right*).

THE ANTERO-INFERIOR ILIAC SPINE APOPHYSIS

A similar force in this same age group may expend itself chiefly in this region Milgram[4] describes such a patient with early limited active hip flexion because of pain. This flexion continued to be restricted after cessation of pain. Roentgenographic studies showed a mass of new-formed bone in the shape of a beak, 2 or more inches long, lying on the anterior hip joint capsule (Fig 8). Attention was called to the apophysis at the tip of this beak that might lead to the mistaken diagnosis of an osteochondroma. The size of the bony mass blocked hip flexion mechanically. Following surgical removal, reattachment of rectus femoris tendon to a higher level, and with cortisone therapy, full recovery of hip motion occurred without further bone formation.

THE ISCHIAL TUBEROSITY APOPHYSIS

Equally rare is injury to this structure; only two cases have been seen. Milch[3] described this entity in great detail recently. Typical is the 18-year-old hurdler who had gradual onset of pain deep in the right buttock, which was his leading leg. This pain was particularly aggravated by the usual preparatory hamstring stretching exercises. During a hurdling event the patient experienced sharp pain on clearing one hurdle and was unable to continue. Swelling, tenderness and discoloration over the right buttock and saddle area were associated with hamstring spasm and weakness. Bed rest with hip extended and with local heat applications caused symptom regression in from 10 to 14 days, after which the patient resumed all activities. When seen 2 years later, the chief symptom was pain in the right buttock on prolonged sitting. The hamstring muscles were strong and without spasm on stretching. Tenderness about right ischial tuberosity was mild. Roentgenographic study demonstrated a crescent-shaped mass of bone, 3 inches long, below and lateral to the ischium, suggestive of an old avulsion of this apophysis (Fig. 9). The patient refused

surgical removal of the free fragment. Clinical reasoning suggests that the major displacement was related in part to the early return to activity; a longer period of protection should favor healing of a clinical entity that probably is much commoner than is recognized now.

CONCLUSION

Injury to the upper femoral epiphysis is commonplace. The disability associated with *a marked displacement is severe; hence, the* physician is alert for early signs of this change. Equally important, however, are other growth centers which doubtless also are injured frequently; being apophyses, i.e., not a joint component, these disabilities are less severe. Protection from repeated injury to a suspected epiphyseal or apophyseal region should further decrease these disabilities.

REFERENCES

1. Gray, Henry: Anatomy of the Human Body, ed. 26, p. 274, Philadelphia, Lea & Febiger, 1954.
2. Howorth, M. Beckett: A Text Book of Orthopedics, p. 695, Philadelphia, Saunders, 1952.
3. Milch, Henry: Traumatic apophysiolysis—a new syndrome, Clinical Orthopaedics No 2, p. *184, 1953.*
4. Milgram, J. E.: Muscle ruptures and avulsions with particular reference to the lower extremities, Am. Acad Orthop. Surgeons Instructional Course Lectures 10:233, 1953.

Lesiones Epiphysee Circa le Articulation Coxal

Summario in Interlingua

Sin reguardo a lor location, le epiphyses es specialmente susceptibile a lesiones. Omne clinicos incontra multe casos de lesiones al regiones de crescentia, specialmente a illos circa le articulation coxal. Lesion del epiphyse supero-femoral es ben recognoscite, probabilemente a causa de su precoce *disturbation del symmetria del gambas.* Per contrasto con isto, le lesiones del altere apophyses circa le articulation coxal non es promptemente recognoscite, con le consequentia de un progressive displaciamento de iste centros per le active functionamento del musculos e le production de massas ossee que pote obstruer le movimento del articulation. Signos clinic de lesion supra un apophyse e de su aggravation per le contraction del musculos attachate a illo indica le *necessitate de reposo absolute pro le struc*turas implicate usque le resanation del tessutos molle es adequate pro prevenir un displaciamento additional.

12

The Effect of Alternating Distracting Forces on the Epiphyseal Plates of Calves; A Preliminary Report*

WILLIAM S. SMITH, M.D.,† AND JAMES B. CUNNINGHAM, M.D.‡

The growth-arresting effect of a compressive type of force across the epiphyseal plate has been well outlined by Haas[5] and others.[1,4] Little is known about the application of the opposite type of force, tension or distraction, across the epiphyseal plate. Volkmann,[9] in 1862, suggested alterations in the growth of long bones as a result of tension and compression on the epiphyseal plate.

In our experiments we sought to determine the effect of the *addition* of a force in the same direction as the already existing force at the epiphyseal plate (Fig. 1). Gelbke[4] utilized a heavy wire attached to an apophysis and to the diaphysis of the adjacent proximal long bone in dogs. Gelbke concluded that "strong and permanent tension does not increase enchondral bone growth; on the contrary it has nearly the same effect as compression."

Figure 2 shows the long-term functional adaptational changes of elongation which have taken place in the apophysis of the greater and the lesser trochanters of two juvenile amputees. Both cases now are adults in whom above-the-knee amputations were performed at the ages of 3 and 6. The extra load required of the iliopsoas and the gluteus medius and minimus produced a situation of continual increased tension on the epiphyseal plates of their respective apophyses at their attachments.

If it can be assumed that the lesser trochanter has been elongated as a result of long-standing tension in the case of the juvenile amputees, then underdevelopment of the lesser trochanter might be anticipated where the influence of the iliopsoas has been removed. Such is the case of poliomyelitis with severe involvement of the iliopsoas in childhood followed into adult life. Figure 3 illustrates the severe underdevelopment of the lesser trochanter in a long-standing case of poliomyelitis. Nevertheless, the effects on endochondral growth exclusive of the epiphysis itself remain in question.

Lacking further clinical cases illustrating the tension phenomenon, attention was turned to the situation of an absence of pressure to an epiphysis where such pressure existed normally.[2] Three separate clinical incidents satisfying this condition were found

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* [illegible] patterned after the coaptator-distractor used for many years by Dr. Carl E. Badgley and was constructed and supplied by courtesy of the Zimmer Manufacturing Co., Warsaw, Indiana.

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occurring in the spine, two from the literature[6,7] and one from our own cases. In all of the cases there was either previous destruction or diminished growth of the anterior vertical height of the vertebral body, either from an old epiphysitis or some other destructive process. So far as the vertebrae above and below the involved body are concerned, this could be regarded as an example of a "lack of normal pressure" phenomenon. Here the anterior portion of the end plates is unopposed, due to the destructive process occurring in the adjacent vertebral body (Fig. 4). Note how the anterior vertical height of the vertebral bodies above and below the involved anteriorly narrowed body is clearly increased There is an unsatisfied pressure deficit. It is also possible that this

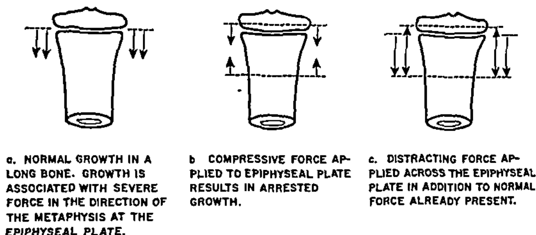


FIG. 1. *(Left)* Normal growth in a long bone. Growth is associated with severe force in the direction of the metaphysis at the epiphyseal plate. *(Center)* Compressive force applied to epiphyseal plate. Results in arrested growth. *(Right)* Distracting force applied across the epiphyseal plate in addition to normal force already present.



FIG. 2. Elongation of the lesser trochanter in adults who had above-the-knee amputation very early in childhood. (Dr. Charles Frantz, Grand Rapids, Mich.)





Fig. 3 *(Left)*. A 31-year-old female who had poliomyelitis at the age of 4. There is no function of the iliopsoas or the hip adductors and only a trace of function of the gluteus medius and minimus.

Fig. 4 *(Right)*. A 30-year-old female with an old deformity of L-2, due presumably to a well-localized epiphysitis. The increased growth of the anterior portions of the adjacent vertebral bodies is easily seen.

effect has followed an abnormal pressure placed upon the posterior portion of the end plates by virtue of the deformity resulting from the diseased vertebrae with unopposed anterior growth.

Another clinical variety of lack of pressure stimulus on an epiphyseal plate may be found in untreated congenital dislocation of any of the major joints. At a glance, congenital dislocation of the hip would appear to fulfill the requirement, but, because of the number of variables, space does not permit further discussion.

EXPERIMENTAL METHODS OF STUDY

Holstein calves were selected for study because of the same advantage noted by Strobino, French and Colonna,[8] namely, the rapid growth rate of the long bones, the large size of the animal facilitating mechanical application of equipment, and the advantage of an isolated epiphysis in the absence of a fibula. Seven animals in all were used. In three animals a type of external fixation was utilized with a ⅛-inch Steinmann pin centrally located in a longitudinal plane parallel to a similar pin in the metaphysis. In the first animals the metacarpal was selected primarily because of the ease of application of the apparatus. By incorporating a turnbuckle apparatus, the distracting force across the epiphyseal plate could be increased easily at daily intervals. Steinmann pins alone were inserted in a similar manner on the opposite side for controls. Two of the animals became infected within 2 weeks, and the third suc-

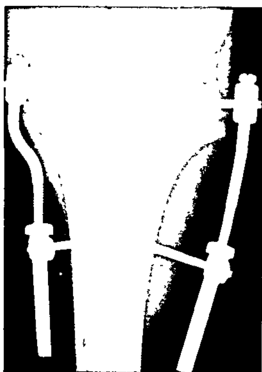


Fig. 5. The apparatus in place. Note the bowing of the pins, indicating the distracting force obtained.



Fig. 6. Autopsy specimen after 30 days. Note the increased height of the proximal tibial epiphysis compared with the opposite side. The epiphyseal plate also is widened. Migration of the pin away from the epiphyseal plate is noted.

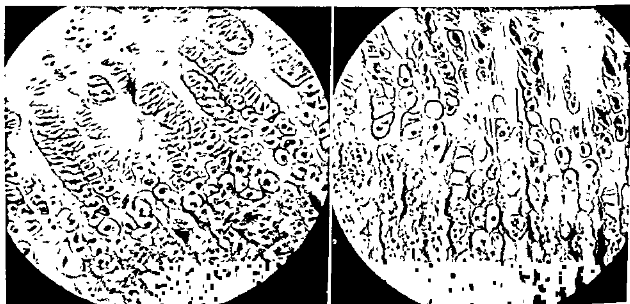


Fig 7. *(Left)* Microscopic section of epiphyseal plate, control. *(Right)* Microscopic section of epiphyseal plate, experimental side.

ceeded in kicking off the apparatus. It was clear that the apparatus was too bulky to employ in a distal epiphysis, and, therefore, subsequent experiments were performed on the proximal tibial epiphysis, a small buried apparatus being used (Fig. 5). Sharp edges were eliminated in this apparatus in order to minimize postoperative trauma at the site of application. Great care was taken to avoid stripping at the epiphyseal plates. At the first application the pins were distracted to a point of moderate bending. The wounds were re-entered at 10- to 14-day intervals in order to increase the amount of tension. Although it was felt that infection was inevitable by this method, nevertheless, two and three re-entries with the application of severe tension could be accomplished.

RESULTS OF STUDY

Of the entire group of animals, the observations reported are based upon the findings in two animals with internal fixation applied at the tibial epiphysis. At each successive examination of the apparatus at 10- to 14-day intervals, no tension was found on the apparatus, indicating that at some time during this period the force associated with growth exceeded the distracting force applied initially. After two re-entries, roentgenographic examination showed a widened epiphyseal plate. At necropsy, epiphyseal separation was expected, but instead a widening of the epiphyseal plate was found (Fig. 6). In one case there was a 3-mm. increase in length in the experimental side as compared with the control. Further examination of the roentgenograms showed that this increase came from an increase in the height of the epiphysis itself This closely parallels the findings in the two juvenile amputees shown previously.

Microscopically a difference was noted between the experimental and the control sides (Fig. 7). On the experimental side the columns of cartilage cells of the epiphyseal plate were large, rectangular and elongated as compared with the closely packed plump cuboidal cells in the control side. Clefts were found between the columns of cartilage on the experimental side.

SUMMARY

1. Clinically, evidence is presented to suggest that alterations in growth of the epiphysis exist as a result of increased tension on the epiphysis or as an absence of tension where this exists normally. No conclusions can be drawn as to the effect of such a force with relation to metaphyseal growth.

2. In two out of seven *Holstein* calves suitable for study, an increase in the width of the epiphysis was noted following the application of an intermittent distracting force at 10- to 14-day intervals.

3. In the latter animals the epiphyseal plate was widened slightly but definitely as compared with the control side.

4. Microscopically the cartilage cells of the epiphyseal plate were elongated where a distracting force was applied at the epiphyseal plate as compared with the control side.

5. Further study, with improvements in apparatus and possibly application to another animal, will be required before it can be concluded that metaphyseal growth is either retarded or accelerated by applying tension to an epiphyseal plate.

REFERENCES

1. Blount, W P., and Clarke, G. R.: Control of bone growth by epiphyseal stapling, J. Bone & Joint Surg. 31-A:464-478, 1949.
2. Bovill, E. G., Jr., Personal communications, 1953.
3. Frantz, C.: Personal communication, 1954.
4. Gelbke, H.: The influence of pressure and tension in growing bone in experiments with animals, J. Bone & Joint Surg. 33-A: 947-954, 1951.
5. Haas, S. L.: Retardation of bone growth by a wire loop, J. Bone & Joint Surg. 27: 25-36, 1945.
6. Hanson, Robert: Some anomalies, deformities, and diseased conditions of the vertebrae during their different stages of development, elucidated by anatomical and radiological findings, Acta chir. scandinav. 60:309-368, 1920.

7. Risser, J.: Important practical facts in the treatment of scoliosis, Am. Acad. Orthop. Surgeons Instructional Course Lectures 5: 257, 1948.
8. Strobino, L. J., French, G. O., and Colonna, P. C.: The effect of increasing tensions on the growth of epiphyseal bone, Surg., Gynec. & Obst. 95:694-700, 1952.
9. Volkmann, Richard: Chirurgische Erfahrungen uber Knochenverbiegungen und Knochenwachstum, Arch. path. Anat. 24: 512-520, 1862.

Le Effecto del Application de Alternante Fortias Distractori a Placas Epiphysal in Vitellos; Reporto Preliminari

Summario in Interlingua

Le arresto de crescentia effectuate per le application de un fortia de typo compressori al placa epiphysal ha essite ben describite per Haas e alteres. Pauco es cognoscite in re le application al placa epiphysal de fortias del typo contrari, i.e., de tension o distraction.

In le presente studio nos ha tentate determinar le effectos del addition de un fortia equidirectional a illo jam existente como resultato del crescentia in le placa epiphysal. Le studio usava un total de septe vitellos de Holstein. Le alterationes del dimension del epiphyse e del placa epiphysal esseva notate insimul con alterationes microscopic al placa.

Supporto clinic del constatationes experimental es citate ab duo casos de amputatos juvenil qui esseva observate posteriormente usque a etates adulte, ab un caso de poliomyelitis a longe durantia, e ab un numero de casos representante certe typos de deformitate vertebral. Le exacte relation inter iste casos clinic e le problema sub investigation es analysate. Le deductiones experimental e etiam le deductiones clinic contribue al explication de certes del alterationes a longe vista con respecto al adaptation functional que es vidite in nostre clinicas.

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The Effect of Juxta-epiphyseal Pyogenic Infection on Epiphyseal Growth*

ROBERT S. SIFFERT, M.D.

The earliest accurate description of the pathogenesis of acute hematogenous osteomyelitis in infancy and in childhood by Starr[29] has been confirmed repeatedly, and the clinical picture has been described time and again.[10,14,18,22,24,30,31,32] It was recognized that the seeding of infection in bone was only one manifestation of a generalized septicemia; treatment, therefore, was primarily that of a sick child and secondarily that of an abscessed part. Green[11] pointed out the difference between the disease in infancy when compared with later childhood and emphasized the benignity of the condition at the earlier age.

With the introduction of antibiotics, the incidence of mortality fell precipitously, and with better control of the foci of infection (skin, respiratory, mastoid, umbilical infections, etc.), which were recognized as the source of the septicemia, the over-all morbidity of acute hematogenous osteomyelitis declined. In the 19 years between 1935 and 1954 the number of deaths each year in the United States from this disease dropped from 1,103 to 8.[19] In spite of this fall in morbidity and mortality, antibiotic therapy alone has not offered the total solution of the control of the disease and its complications. The resistance of the organism, the degree of general toxemia, the amount of bone and soft-tissue involvement, the age of the child and the anatomic location of the disease process are all factors that must enter into the clinical judgment as to whether or not surgical intervention to evacuate subperiosteal, soft tissue or joint abscesses is indicated.[1,4,14,26] Attention also must be paid to intelligent general orthopaedic care, such as proper immobilization to prevent further local spread of the infection, to avoid joint dislocations, particularly in hip involvement in infants, and to prevent pathologic fractures, epiphyseal slips and other complications of the disease.[11]

During the last few years the experiences of antibiotic therapy in acute hematogenous osteomyelitis have been evaluated.[2,5,15] In general, they indicate that there has been an alarming and steady increase in the resistance of a large number of strains of bacteria to the antibiotics, making more difficult the treatment of established bone infections, as well as foci of infection elsewhere that are potential sources of hematogenous spread to bone. The staphylococcus, which is most prone to develop resistance to the antibiotics,[5] is the organism involved most commonly in acute hematogenous osteomyelitis in children over the age of 2.[12] Some observers feel that the staphylococcus accounts for a large percentage of bone infections in infancy as well,[3,16] while others have reported that either the streptococcus[7,12,26] or a great variety of organisms[13] may be in-

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volved as the etiologic organisms during this early period of life.

The antibiotics are reported to have made little or no difference in the incidence or the course of acute hematogenous osteomyelitis in infants under 1 year of age.[3] In fact, it has been suggested that an actual increasing incidence in the disease might be expected in the future, since more infants are surviving bouts of overwhelming septicemia because of the antibiotics, and a certain percentage of these will have residual bone infections as a result of hematogenous seeding of resistant bacteria to bone.[23]

In the differential diagnosis of a painful extremity, the clinician must be aware of the changing character and behavior of various organisms, grown partly or completely resistant to the antibiotics, creating a totally different picture of the disease. It is possible, therefore, to develop acute or masked subacute bone infections via the hematogenous route, even though the patient is being treated with antibiotics for a primary focus of infection or septicemia. Rather than the classic picture of primary acute septicemia followed by metaphyseal localization, metaphyseal tracking with cortical rupture and subperiosteal abscess formation, osteomyelitis developing under antibiotic therapy and due to resistant organisms, may result solely in areas of sharply localized bone destruction and abscess formation. Symptoms then may be insidious in onset, or the lesion may become walled off and remain quiescent, only to flare up after antibiotics have been stopped or at a later period corresponding to an increase in virulence of the encapsulated organism or decrease resistance of the host. Trueta[30] emphasized the variability in potential activity of the implicated organism with its antibiotic resistance and the variability in severity and clinical picture of the disease in different geographic locations. Gilmour, in Australia,[9] noted that whereas 10 years ago the disease had been well controlled by penicillin, since 1950 it was virtually the same as it had been before penicillin, due to the increasing prevalence and changing character of the staphylococcus, the increase in incidence in young children and the increase in severity of the disease.

In general, the seeding of bacteria during a bout of septicemia may occur in almost any organ. Bacterial growth localizes and gains a foothold in the metaphyseal area of a long bone, apparently primarily because of the slowed circulation in the metaphyseal sinusoids[30] in the presence of degenerating elements of epiphyseal cartilage. Fixation of infection in these areas by the rich reticuloendothelial activity, lack of phagocytic activity and trauma with formation of small intra-osseous hematomas have been implicated,[1,6,14] but for the most part all are conjectural. Once a small metaphyseal abscess has formed, the amount of future involvement depends upon a large number of variable factors. These include the location of the lesion, the age of the child, the organism involved and its virulence, host resistance and susceptibility, antibiotic resistance and the time that therapy was begun Green and Shannon[12] demonstrated that in infants and in children below the age of 2, the disease was of brief duration with rapid local healing, infrequent sequestration and rare recurrences, whereas over this age the disease was of longer duration with slow healing, a tendency toward sequestration, sinus formation and residual sclerosis with frequent recurrences.

THE EFFECT OF LOCAL PYOGENIC INFECTION ON EPIPHYSEAL GROWTH

The large sinusoidal channels, loose metaphyseal cancellous structure, thin cortex and loosely attached periosteum of infancy permit early decompression of an intra-osseous abscess, with tracking of the pus into the subperiosteal area, local soft tissues or adjacent joint.[11] Therefore, although there is relatively little local metaphyseal bone destruction, this process predisposes to the two most common complications affecting epiphyseal growth in infancy, namely, pyogenic arthritis and epiphyseal slipping.

Acute Pyogenic Arthritis

Entrance of pus into an adjacent joint generally occurs in those areas where the epiphyseal plate is partly (knee, shoulder) or wholly (hip) surrounded by capsule and, therefore, partly or wholly intra-articular.[14,24] Drainage of the metaphyseal abscess occurs directly into the joint. It is uncommon to observe rupture of a subperiosteal abscess through the dense periosteal attachment at the epiphyseal plate in those areas where the plate is extra-articular. It has been suggested that occasional pyogenic arthritis might develop as a result of epiphyseal slips[17] or direct extension along infected lymphatics, thrombi and, possibly, local cellulitis associated with the pyogenic process in the metaphysis and subperiosteal areas.[1,32] In instances of secondary pyogenic arthritis, the infection usually is virulent and resistance low, since it is a complication of a well-established rapidly tracking metaphyseal abscess. The hip is the joint involved most commonly in infancy, and the degree of destruction of the capsule and the epiphysis itself bears a direct relationship to the virulence of the organism, the stage at which therapy is begun (aspiration and/or surgical drainage, as well as antibiotic therapy) and the resistance of the organism to the antibiotic used. Virulent attack upon the joint in infancy and in early childhood usually results in partial or complete destruction of the epiphysis and the epiphyseal plate, along with its growth potential (Figs. 1 & 2). It is generally agreed that a large number of cases of apparent primary pyogenic joint involvement are



Fig. 1. *(Left)* Roentgenogram of the hip of a 1-year-old child following acute hematogenous osteomyelitis of the upper femur with involvement of the hip joint. The femoral capital epiphysis has been destroyed completely, along with the capsular and surrounding extracapsular structures, resulting in dislocation of the hip. *(Right)* Roentgenogram of the same patient at the age of 4, demonstrating the deformity resulting from the destruction of the femoral capital epiphysis, as well as the extra-articular apophysis of the greater trochanter.

truly instances of small localized and unrecognized foci of bone infection that have extended into the joint.[1,7,10,17,20,25]

Probably because of the small amount of ossification of the infant pelvis and the large amount of cartilage making up the acetabulum, bony ankylosis in infancy does not occur.[33] However, there may be capsular destruction, with rupture of purulent material into the surrounding soft tissues. If decompression occurs spontaneously by this or by surgical means, the epiphysis itself may be spared partly or completely, but later epiphyseal growth disturbances, secondary to dislocation, subluxation, acetabular irregularity or aseptic necrosis, with resultant deformity, may be observed.

In the older child, the relatively thin articular cartilage is more vulnerable, and bony ankylosis is not an uncommon sequel to secondary pyogenic arthritis. Infections of sufficient virulence to cause bony ankylosis usually result in destruction of some portion of the epiphyseal center and the growth potential of the epiphyseal plate (Fig. 3).

Epiphyseal Slipping

The confinement of bone destruction to the metaphyseal area, with local cortical perforation and periosteal stripping, may produce a mechanical loosening of the epiphysis.[32] In spite of attempts at adequate immobilization, slipping of the epiphysis, often with concomitant septic arthritis, may occur. In simple epiphyseal slips, where the infection comes under control either by surgical drainage of the abscess or effective antibiotic therapy, without further damage to the normally resistant epiphyseal cartilage or joint surfaces, the bone usually heals without sequestrum, and the only residual deformity is the tilt of the epiphysis equal



Fig. 2. *(Left)* Roentgenogram of the hip of a 1-year-old child following pyogenic involvement of the upper femur, where the infection has been confined to the hip joint without involvement of the extra-articular structures. *(Right)* Roentgenogram of the same patient at 6 years of age. The femoral capital epiphysis has been destroyed, while the extra-articular uninvolved apophyses of the greater trochanter and the lesser trochanter have continued to grow, causing this characteristic deformity.

to the degree of original slipping. Since slipped epiphysis due to juxta-epiphyseal pyogenic infection is almost exclusively a complication of infancy or of early childhood, complete remodeling usually occurs.

Involvement of the Epiphyseal Plate and the Epiphysis

Because of the extreme resistance of the epiphyseal plate to infection, only occasionally does a virulent infection involve the plate directly. The bone abscess in a child of any age usually comes to rest against the epiphyseal plate,[29] and local inflammatory changes can be seen in that area of the metaphysis where destruction of cartilage cells is occurring as the final stage in the physiologic process of enchondral ossification. These changes do not affect epiphyseal growth itself, since the proliferating and the palisading cells, which are responsible for initiation and maintenance of epiphyseal growth, are on the epiphyseal side of this avascular resistant barrier. Occasional temporary stimulation of growth occurs, due probably either to hyperemia of the local inflammation or thrombosis of the medullary vessels.[30] Wilson and McKeever[34] noted that growth stimulation was related more commonly to diaphyseal than to juxta-epiphyseal foci of infection. When the infection is localized eccentrically beneath the epiphyseal plate, eccentric stimulation and angular deformities may occur, particularly in the lower femoral and the upper tibial epiphyses, with the production of genu valgum. Stimulation ceases when the inflammatory phase is over and osseous vascularity is restored to normal. Permanent discrepancies in actual bone length due to epiphyseal stimulation, where there is no actual destruction of the plate, have not been noted.[30]

In the young child the abscess is confined to the metaphysis for a brief period before egress is found into the subperiosteal area, the soft tissues or the joint. Epiphyseal destruction usually occurs as a result of overwhelming secondary pyogenic arthritis[8] or



Fig. 3. Roentgenogram of an unusual case of pyogenic infection of the upper femur in childhood with joint involvement, ankylosis and complete destruction of growth from the upper femoral epiphysis. A draining sinus of 30 years' duration was explored, and a sequestrated intact femoral head, the size of that of a child of 6, was removed from the base of the sinus.

local abscess formation. Primary epiphyseal foci, direct extension of the infection from the metaphysis into the epiphyseal plate and secondary pyogenic arthritis resulting from extension of a metaphyseal abscess through the epiphyseal plate into the epiphysis and then into the joint are all rare.[20]

In older children there is a distinct difference in the complicating effects of juxta-epiphyseal infection on epiphyseal growth. The bone itself is larger, and the metaphysis consists of a more complex and denser trabecular pattern. Therefore, a small localized abscess cannot expand within the metaphysis as easily as it can within the bone of an



FIG. 4. *(Left)* Roentgenogram of a 1-year-old child who developed acute *Staphylococcus aureus* involvement of the lower medial femur during the first few weeks of life, with apparent complete destruction of the medial half of the lower femoral epiphysis. The genu varum deformity has resulted from continued normal growth from the uninvolved lateral half of the epiphysis *(Right)* Roentgenogram of the same patient at the age of 8, demonstrating an amazing degree of restoration of the architecture of the knee. Spontaneous correction of the genu varum has occurred by growth of the medial peripheral portion of the lower femoral epiphysis, which was cartilaginous at 1 year of age *(left)*, when it appeared as though this section had been destroyed completely. At that time osteotomy designed to correct the genu varum would have been an unnecessary procedure and might have resulted in secondary genu valgum due to later normal growth of the medial section of the epiphysis



infant. There is local necrosis of bone by edema, thrombosis, pressure and the destructive effect of bacterial toxins, with the formation of a metaphyseal abscess. The abscess may become completely resolved under antibiotic therapy with no effect on the epiphyseal plate, or may remain as a

FIG. 5 Roentgenogram of the knee in a 10-year-old child complaining of local pain for one year. A juxta-epiphyseal abscess has extended to involve a localized central portion of the epiphyseal plate, resulting in a serpentine abscess channel in the metaphysis as normal growth continued. There was no leg-length inequality or angulation at the knee. At operation *Staphylococcus aureus* was recovered from the abscess, and, in spite of central curettage of the epiphyseal plate, no disturbance in growth occurred.

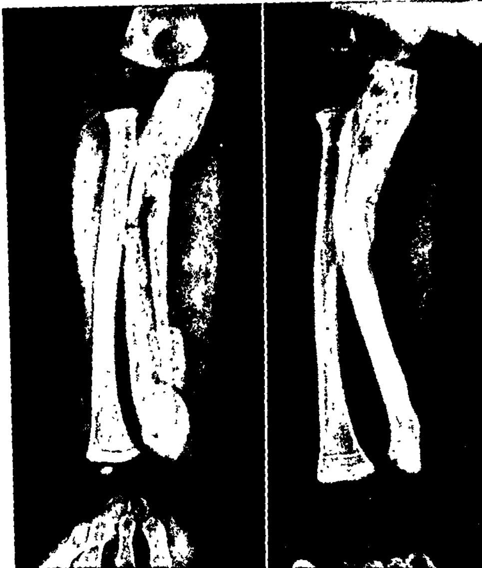


FIG. 6. *(Left)* Roentgenogram of the forearm of a 2-year-old child with *Staphylococcus aureus* infection of the upper end of the ulna and secondary involvement with periosteal reaction along the entire shaft of the bone. There is an angulated pathologic fracture of the ulna shaft and of the involucrum. *(Right)* Roentgenogram of the forearm in the same child taken 6 months later, demonstrating healing of the fracture with residual angular deformity at the fracture site. A secondary curve of the lower ulna, due to compensatory epiphyseal growth has occurred in order to maintain the normal integrity and alignment of the wrist joint. As remodeling of the fracture angulation occurs, associated straightening of the lower compensatory angulation will also take place.

well-walled-off Brodie's abscess growing away from the plate as normal growth continues. However, virulent infections, locally confined because of mechanical resistance to spread, often result in adjacent epiphyseal plate and epiphyseal involvement. Frequently the amount of irreparable damage done to the epiphyseal plate cannot be determined at the time of the original infection, since much of the involved structure in the younger child is cartilaginous and does not show up on roentgenologic examination. Often it is surprising to note the degree of apparent regeneration of bone-growth potential, which in reality represents areas never completely destroyed (Fig. 4). Therefore, deferment of reconstructive and stabilizing procedures, particularly at the hip, should be emphasized until adequate time has elapsed to assess the actual degree of damage suffered by the growth center.

The amount of growth disturbance is directly related to (1) the total area of the cartilage plate destroyed in relation to the total size of the plate and to (2) the actual anatomic location of the destroyed area in the plate. In these respects the effect of destruction of areas due to pyogenic infections is similar to the effect of traumatic injuries to the epiphyseal plate. Centrally involved areas of small degree, like transepiphyseal curettings or local central epiphyseodesis due to transepiphyseal nails or wires, have little or no effect on epiphyseal growth[21,27,34] (Fig. 5).

Larger central defects may retard growth,

but usually there will be no angular deformity, since there is even and concentric growth from the uninvolved peripheral portions of the epiphyseal plate. However, eccentric or peripherally destroyed areas of the plate, with or without involvement of the adjacent epiphyseal bone, when large enough, will cause eccentric epiphyseodesis and result in increasing angular deformity and shortening.

Secondary Effects on Epiphyseal Growth

Pathologic Fracture. A less important complication occurring usually in early childhood is that of fracture of the shaft of the involved bone through an area weakened by extension of the juxta-epiphyseal infection to the subperiosteal area and diaphysis. Malalignment causing secondary abnormal pressures on the epiphyseal plate of the fractured, and often the adjacent articulating, bone as well often results in gross deformities. Since this complication is most common in the younger child, deformities of this nature, where there is no actual destruction of the epiphyseal plate, usually are resolved by periosteal remodeling and minor compensatory alterations in epiphyseal growth, so that little or no permanent abnormality exists (Fig. 6).

Effect on the Epiphysis of Companion Bones. Juxta-epiphyseal involvement of the lower tibia in the presence of a healthy fibula, or similar involvement of the lower radius with a healthy companion bone, is not uncommon Simple stimulation of growth due to subepiphyseal hyperemia or diaphyseal extension is associated almost invariably with concomitant increase of the companion bone with no deformity. However, when virulent infection results in local cessation or retardation of epiphyseal growth in one bone, the rate of growth in the uninfected companion bone usually is either not retarded or actually is accelerated. The continued growth of the healthy bone, then, may produce a "pulsion" force[34] which exerts angular pressure on the end of the involved bone, resulting in angular deformities at the epiphyseal plate and alterations in alignment of the articular surface.[28]

SUMMARY

A brief outline is presented describing the more common effects of juxta-epiphyseal pyogenic infection on epiphyseal growth.

It is suggested that the universal security felt initially with the introduction of the antibiotics in the treatment of acute pyogenic bone infections in children be tempered, and that the disease be considered seriously in the differential diagnosis in all cases of painful lesions in the extremities of children in the light of increasing bacterial resistance to the antibiotics and the associated changing clinical picture of acute hematogenous osteomyelitis.

REFERENCES

1. Bisgard, J. Dewey: The relation of pyogenic arthritis to osteomyelitis. Surg., Gynec. & Obst. 55:74-80, 1932.
2. Blair, John E.: Microbial complications of antibiotic therapy, Bull Hosp. Joint Dis. 17:284-296, 1956.
3. Blanche, Donald W.: Osteomyelitis in infants, J. Bone & Joint Surg. 34-A:71-85, 1952.
4. Brown, Henry P., Jr.: Acute hematogenous osteomyelitis of the long bones, Ann. Surg. 109:596-614, 1939.
5. Bryer, Morton S.: The chemotherapy of bacterial infections refractory to the common antibiotics, Am. J. Med. 18:782-791, 1955.
6. Dickson, Frank D.: The clinical diagnosis, prognosis and treatment of acute hematogenous osteomyelitis, J.A.M.A. 127:212-217, 1945.
7. Einstein, Robert A. J., and Thomas, Colin G , Jr.: Osteomyelitis in infants, Am. J. Roentgenol. 55:299-314, 1946.
8. Fraser, John: Acute osteomyelitis, Brit. M. J. 22:605-610, 1924.
9. Gilmour, W.: Acute osteomyelitis in children: abstract of annual general meeting of Australian Orthopedic Association, J. Bone & Joint Surg 38-B:596, 1956.
10. Green, Morris, Nyhan, William L., and Fousek, Mildred: Acute hematogenous osteomyelitis, Pediatrics 17:368-382, 1956.

11. Green, William T.: Osteomyelitis in infancy, J.A.M.A. 105:1835-1839, 1935.
12. Green, William T., and Shannon, J. G.: Osteomyelitis of infants: a disease different from osteomyelitis of older children, Arch. Surg. 32:462-493, 1936.
13. Greengard, Joseph: Acute hematogenous osteomyelitis in infancy, M. Clin. North America 30:135-145, 1946.
14. Hart, Vernon L.: Acute hematogenous osteomyelitis in children, J.A.M.A. 108:524-528, 1937.
15. Howe, Chester W.: Prevention and control of postoperative wound infections owing to *Staphylococcus aureus*, New England J. Med. 255:787-794, 1956.
16. Key, J. Albert: Discussion of [3].
17. Lewis, Dean: Acute osteomyelitis, J.A.M.A. 92:783-786, 1929.
18. Ober, Frank R.: Osteomyelitis in children, Am. J. Surg. 39:319-326, 1938.
19. Personal Communication: Children's Bureau, Department of Health, Education, and Welfare, Social Security Administration, Washington, D. C.
20. Phemister, D. B.: The pathology and treatment of pyogenic arthritis, Pennsylvania M. J. 32:52-57, 1928.
21. ———: Discussion of [34].
22. Pierson, J. W.: The roentgenology of osteomyelitis, J.A.M.A. 126:884-886, 1944.
23. Potter, C. M. C.: Osteomyelitis in the newborn, J. Bone & Joint Surg. 36-B:578-583, 1954.
24. Reich, Rudolph S.: Purulent arthritis, J. Bone & Joint Surg. 10:554-578, 1928.
25. Robertson, Robert C.: Acute hematogenous osteomyelitis, J.A.M.A. 107:1193-1200, 1936.
26. Self, Edward B.: Acute hematogenous osteomyelitis, Pediatrics 1:617-626, 1948.
27. Siffert, Robert S.: The effect of staples and longitudinal wires on epiphyseal growth, J. Bone & Joint Surg. 38-A:1077-1089, 1956.
28. Speed, Kellogg: Growth problems following osteomyelitis of adolescent long bones, Surg., Gynec. & Obst. 34:469-476, 1922.
29. Starr, Clarence L.: Acute hematogenous osteomyelitis, Arch. Surg. 4:567-587, 1922.
30. Trueta, J.: Acute hematogenous osteomyelitis: its pathology and treatment, Bull. Hosp. Joint Dis. 14:5-23, 1953.
31. Wade, R. B.: Acute osteomyelitis in children, M. J. Australia 1:264-268, 1929.
32. Wilensky, Abraham O.: Osteomyelitis: its pathogenesis, symptomatology, and treatment, New York, Macmillan, 1934.
33. Wilkinson, F. R.: Discussion of [3].
34. Wilson, John C., and McKeever, Francis

Le Effecto de Juxta-Epiphysal Infectiones Pyogene Super le Crescentia del Epiphyse

Summario in Interlingua

Le introduction de antibioticos in le tractamento de affectiones pyogene de ossos ha grandemente reducite le morbiditate de infectiones chronic. Tamen, le staphylococco —que es le organismo le plus frequentemente implicate—es etiam le organismo con le plus forte tendentia a devenir resistente contra le antibioticos. Le resultato es que (1) pauc differentia es a notar in le incidentia e le curso de acute osteomyelitis hematogene in infantes, durante que (2) un alteration marcate ha occurrite in le tableau clinic de acute infectiones ossee con focos de resistentia que se disveloppa durante le therapia a antibioticos.

Infectiones juxta-epiphysal in infantes ha lor plus grande effecto destructive super le placa de crescentia in le presentia de un complicante arthritis pyogene. Del altere latere, dislocate epiphyses que non es associate con virulente affectiones articular, resulta usualmente in pauc o nulle deformitate. Quando in juveniles de etate plus avantiate, le infection es satis virulente pro superar le resistentia natural del placa epiphysal, varie grados de arresto epiphysal pote occurrer. In isto le resultante deformitate depende del quantitate total e del loco del destruite cartilagine epiphysal.

14

Effects of Trauma Upon Epiphyses

William N. Harsha, M.S., M.D.*

The purpose of this chapter is to review some better-known facts of epiphyseal growth and their relationship to traumatic disturbances as seen in a common clinical practice. I will relate this review to some recent investigative studies and, less frequently, to used therapeutic technics.

As the skeleton matures from its fetal period to completion of growth, the epiphyses play the most important part in regulating linear growth and the contour of bone ends. As a corollary, abnormality of epiphyseal growth affects bone length or joint contour, modifying the walking patterns, cosmetic appearances and the ability properly to use the extremities.

For the purpose of this chapter, trauma may be defined as a force tending to shear, compress or distract a segment of the body unduly. Such trauma may consist of a single acute blow or many small repeated insults

SPRAINS

The medical literature is remarkedly devoid of dissertations dealing with sprains and their sequelae. The orthopaedist is all too familiar with the laissez-faire attitude of many practitioners toward sprains of ankle, knee or wrist. This attitude is even more manifest in dealing with similar injuries of childhood. A sprain is the result of a force that tends to pull apart the ligamentous supporting structures of a joint or fibrous insertions of a tendon. Most joint capsular fibers attach on the epiphyseal side of the epiphyseal plate. Structurally, the metaphyseal aspect of the epiphysis is weakest, and disruption of the epiphyseal plate is a real possibility. It has been shown that the strength of the fibrous capsule is from two to five times greater than that at the metaphyseal-epiphyseal junction.[5,8] Many authors believe that it is possible to disrupt the epiphysis completely from its metaphyseal face and yet not displace it, owing to the balanced muscular, periosteal and perichondrial anchors.

Frequently the orthopaedist, interested in athletic injuries, finds that carefully taken roentgenograms of so-called sprains in the child, particularly about the ankle, show not disruptions of ligamentous structures but actually periosteal tears, together with partial separations at the metaphyseal-epiphyseal junction, yet without loss of anatomic position.[13]

Supporting this thesis, the so-called simple sprain in a growing child with open epiphyses merits a careful roentgenographic examination and therapeutic care. We see a large number of growing children who have developed an asymmetric plane at the knee joints. On close questioning they can recall trauma to the knee that was interpreted as a sprain. The possibility that these sprains represented a partial disruption at the metaphyseal-epiphyseal junction must be considered. These areas are devitalized temporarily, allowing the opposite side of the epiphysis to continue its growth. An irregu-

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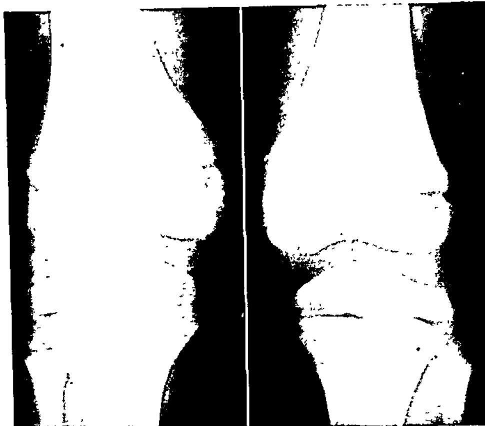


FIG. 1. Nine-year-old boy. Strain injury to right knee with pain along inner aspect at 6 years of age. No treatment. At 9 years of age there is 10° greater varus deformity of the right knee than the left. Is this retardation of medial femoral or tibial epiphyses due to trauma?

larity of growth of that part of the joint usually occurs. These asymmetries of joint lines generally are minimal, yet they may be of such magnitude as to predispose to future arthritic changes.

It is well known that a fracture, including cartilage, bone or the metaphyseal-epiphyseal plate, is subject to slow union or nonunion if movements are permitted. This is especially true if motion is superimposed upon normal or abnormal stresses such as weight-bearing The same type of injury not subjected to weight-bearing or undue motion heals regularly. It re-establishes its circulation and for all practical purposes is indistinguishable from its counterpart. An example of this is seen in slipped capital femoral epiphysis where, if kept moving under weight-bearing, a marked deformation of the femoral head takes place.

The author believes that all sprains of weight-bearing joints in children must be considered to be severe injuries. He prefers to immobilize them in plaster, usually carrying them non-weight-bearing for from 7 to 14 days. Then, continuing the immobilization, weight-bearing is permitted for an additional 7 to 14 days. Periodic roentgenograms during the entire growth period are routine procedures. Any irregularity of epiphyseal growth at the injured area will be detected. If it develops, proper care is indicated. The author believes that it is reasonable to treat a child who is developing a varus or a valgus deformity of the knee by the application of counterposing shoe wedges or braces. More severe cases require arresting epiphyseal staples properly inserted.

Case 1 (Fig. 1). A 9-year-old boy first was seen after complaining of pain along the inner side of his right knee which had become progressively more severe during the preceding 9 months At times it was severe enough to cause him to limp. There never was swelling or deformity of the joint. The mother did recall a specific instance, at 6 years of age, when the boy jumped from a tree, twisting his right knee. He had some swelling and soreness on the medial aspect. The family doctor stated that it was a sprain and that he should run it off. No further treatment was given Roentgenograms at our first contact revealed 10° more varus of the right knee than of the left. This is entirely consistent



FIG. 2. An 11-year-old boy with a posterolateral epiphyseal displacement of right ankle. Roentgenograms 2 years later show valgus deformity due to asymmetric arrest of distal tibial epiphysis.

with diminution of epiphyseal growth of the medial femoral condyle for an indefinite period before it continued at the same rate as the lateral condyle. Such asymmetric weight-bearing is a well-known predisposing factor in degenerative changes

This boy was treated with a medial heel and a sole wedge. At the end of 4 months he had gained a significant degree of symptomatic relief. It is anticipated that his roentgenograms will show a tendency toward correction of his varus deformity. If the varus deformity were more severe, there would be no hesitancy whatever about stapling the medial femoral condyle until growth had equalized.

EPIPHYSEAL DISRUPTIONS

The same type of trauma that produces a strain, if more severe in character, will separate the epiphysis. Usually a small triangular piece of cortex at the margin of the epiphysis is fractured. One margin of the epiphyseal plate is subjected more to compression than to a shearing force. A shearing force is rarely applied alone to any unit of the body. Usually trauma is a combination of a torsional and a compressive force. In epiphyseal-metaphyseal fractures, the blood supply to the epiphysis from the metaphysis is completely divided. The growth is stopped in that part of the epiphysis that faces the metaphyses. This period of growth cessation is proportional to the degree of trauma and separation. In situations where a substantial triangular piece of metaphysis remains attached to the epiphysis, circulation to that area remains intact. Growth will continue at this point but cease at other areas of the epiphysis where separation occurs.*

Epiphyseal fractures may occur at the distal end of the radius and the distal and the proximal ends of the tibia.

Case 2 (Fig. 2). While playing football, an 11-year-old boy twisted his right ankle and had immediate pain and swelling. Roentgenograms revealed a posterolateral displacement of the metaphyseal-epiphyseal junction with the triangular portion of the anteromedial tibial cortex included. This was reduced under anesthesia and held by non-weight-bearing plaster for 4 weeks. A stable union of the metaphyseal-epiphyseal junction resulted. Roentgenograms at 6 months showed a satisfactory alignment and no apparent growth irregularity. Roentgenograms 1½ years later showed a slight valgus deformity of the talotibial joint and at 2½ years a severe valgus of the tibial mortise.

These findings demonstrate that the metaphyseal-epiphyseal area on the posterior aspect of the tibia was completely divested

* The editor cannot agree with this statement but holds that every writer is entitled to express his opinion.



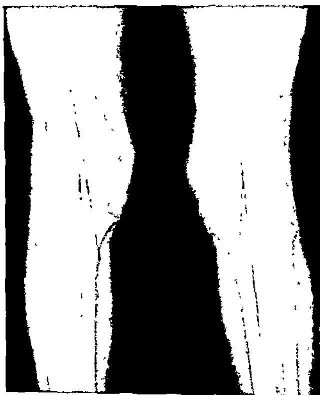


Fig. 3. *(Top, left & right)* A displaced distal femoral epiphysis, fixed with crossed Kirschner wires after two unsuccessful attempts at closed manipulation and plaster immobilization. *(Bottom)* Roentgenogram taken 2 years later; no evidence of asymmetric growth.

of its apposition with the metaphysis. Its growth potential was disturbed for a period substantially longer than that portion of the epiphysis attached to the triangular metaphyseal fragment of bone. Certainly no criticism of the treatment in this could be made, but it is important for the orthopaedist to recognize this possibility.

RECURRENT DISPLACEMENTS OF EPIPHYSES

These unstable disruptions require fixation to prevent recurrence. There are a number of situations where the fracture dislocation has been reduced, though improperly immobilized. Recurrent slipping will occur. Notable in this category is the slipped capital femoral epiphysis. There may be a primary metabolic defect that makes the metaphyseal-epiphysis junction more labile to stress. Many in their early teens who sustain sufficient injury may slip the capital femoral epiphysis. Otherwise they do not present stigmata of a metabolic disorder. Regardless of the etiology, an acute slipping of the capital femoral epiphysis may be reduced by manipulation followed by nailing. This is accepted rather generally as the best form of treatment and the only epiphysis so regarded.

Recurrent slipping also occurs at the distal end of the tibia, the distal end of the femur and, more often, the distal end of the radius.[1] Open reduction with internal fixation of these lesions is not the usually accepted treatment. There is a tendency to accept from 10° to 50° angulation in the expectation that the body will correct such



FIG. 4. *(Left)* An epiphyseal displacement of distal left radius, fixed with crossed *Kirschner wires after unsuccessful attempt at closed reduction. (Right)* Roentgenograms taken 2 years later; no evidence of growth disturbance.

an angulation in due time. If this angulation occurs in a young child and is in the plane of motion of the joint, remolding of the epiphysis will occur, and open surgery is contraindicated. This has been emphasized by Watson-Jones[14] and Blount.[4] The author is entirely in accord with this. There are instances of gross displacement in which even partial alignment cannot be achieved and held by a closed method. Epiphyseal fractures that occur in later childhood do not permit an ample growing time for reconstruction. Inevitably such cases result in deformities. When instability exists, the fracture may be held by crossed Kirschner pinning of the epiphysis.

Case 3 (Fig. 3). A 4-year-old boy fell from his tricycle and sustained a fracture dislocation of the inferior femoral epiphysis with anterior displacement of the distal fragment. This fracture was reduced under general anesthesia and immobilized in plaster. Forty-eight hours later it slipped back to its original position It was again reduced, and the boy was placed in a hip spica with the knee flexed 45°. Reduction was not maintained. A third anesthetic was required, and the fracture dislocation was stabilized by crossed Kirschner wires Two years later no evidence of growth disturbances existed.

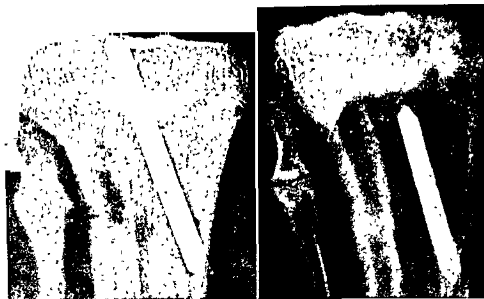
Case 4 (Fig. 4). An 11½-year-old girl fell from a water tank on her outstretched right forearm, sustaining a dislocation of the distal radial epiphysis. This was reduced, and a cast was applied. Within 12 hours the deformity had recurred. It was remanipulated and pinned blindly with Kirschner wires The wires were removed in 4 weeks. The patient has been followed 2 years without evidence of growth disturbances.

Unstable epiphyseal fractures are indications for Kirschner wire fixation. A more assured holding of the fracture in a satisfactory position becomes possible. To bind the joint in forced abnormal positions that tend to compromise the circulation, stretch ligaments and tendinous supports is not desirable.

SURGICAL TRAUMA

Deliberate surgical trauma injures epiphyses. However, in recent years it has been shown that some trauma can be imposed upon epiphysis without altering the contour of bone ends. It may be necessary to arrest epiphyseal growth completely in some cases. Stripping of the periosteum about the edges of the metaphyseal-epiphyseal junction, if complete, will arrest the epiphysis.[2] Phemis-

FIG. 5. Showing placement of metallic rod across the center of epiphyseal plate of experimental rabbits and demonstrating the extrusion of the rod as epiphysis grows in asymmetric manner. (Siffert, R. S: J. Bone & Joint Surg. 38-A:1077)



ter[10] and others have demonstrated that reversing mortises of cortical bone crossing epiphyseal lines will produce epiphyseal arrest. Any surgical procedure that might destroy the epiphyseal plate is fraught with danger. Open reduction of fracture dislocations of epiphyses is a procedure to be carried out with particular care but is preferable to accepting irreversible deformities. Passing a large metallic nail through the mid-portion of a growing epiphysis will destroy that section of the epiphysis of which the object occupies but does not destroy the remaining portions.[6,11] The epiphysis will continue to grow and in time will extrude the nail (Fig. 5).

Relying on this type of experiment, more orthopaedic surgeons should be able to transfix epiphyses with fixing devices. Closed reductions of fracture dislocations at the metaphyseal-epiphyseal junction may be fixed with cross Kirschner wires without danger Better results will be achieved. The necessity for repeated anesthesias and manipulative trauma will be avoided.

Blount,[3] Haas[7] and others have demonstrated that it is possible to place metallic staples across the peripheral epiphyseal lines and arrest their growth for varying lengths of time.[9] Complete arrest is possible if they are left in place long enough. This occurs by creating great compression at the epiphyseal-metaphyseal zone.

Colonna[12] and his co-workers have shown that there is a critical degree of compression in which epiphyseal growth will cease, and, if this compression is maintained for a sufficient period of time, complete epiphysiodesis may occur. The amount of compression necessary is equal, roughly, to three times the body weight. The current metallic staples used in epiphyseal stapling are those suggested by Blount which, in the author's hands, have required about 450 pounds of traction to spread.

SUMMARY

1. The metaphyseal-epiphyseal junction in growing children is more labile to partial and even complete disruption by shearing or compressing forces than are the ligamentous structures supporting a joint. A plea is made for adequate roentgenograms at the time of injury and, more important, for adequate follow-up of all minor sprains and nonfracture injuries about joints of growing children. Only by a roentgenographic follow-up can irregularity of the joint contour be demonstrated and proper steps be instituted early enough to reverse the tendency.

2. It is possible and practical to cross

central portions of the epiphyseal-metaphyseal junctions with smooth metallic wires, fixing fracture dislocations of the epiphysis easily and securely. This produces far less trauma than repeated forceful manipulations and maintenance in extreme positions during healing.

3. It has been shown that epiphyseal growth can be diminished by bridging the metaphyseal-epiphyseal junction with staples. Usually this is temporary, but it will become irreversible if appositional bone forms across the epiphyseal line. Complete arrest of the epiphysis is accomplished by altering the arrangement at the metaphyseal-epiphyseal margins so that appositional bone growth will occur.

REFERENCES

1. Aitken, Alexander P.: Further observations of the fractured distal radial epiphysis, J. Bone & Joint Surg. 21:922, 1935.
2. Blount, W. P.: Fracture in Children, Baltimore, Williams & Wilkins, 1955.
3. Blount, W. P., and Clarke, G. R.: Control of bone growth by epiphyseal stapling, J. Bone & Joint Surg. 31A:464, 1949.
4. Blount, W. P., and Zeier, Frank: Control of bone length, J.A.M.A. 148:451-457, 1952.
5. Fairbanks, T.: An Atlas of General Affections of the Skeleton, Baltimore, Williams & Wilkins, 1951.
6. Ford, L. T., and Key, J. A.: A study of experimental trauma to the distal femoral epiphysis, J. Bone & Joint Surg. 38A:84, 1956.
7. Haas, S. L.: Retardation of bone growth by a wire loop, J. Bone & Joint Surg. 27:25, 1945.
8. Hutchinson, Jonathan, Jr.: Lectures on injuries to the epiphyses and their results, Brit. M. J. 1:673, 1894.
9. Myerding, H. W.: Giant cell tumors of bone, J. Internat. Coll. Surgeons 27:414, 1952.
10. Phemister, D. B.: Operative arrestment of longitudinal growth of bones in treatment of deformities, J. Bone & Joint Surg. 15:1, 1933.
11. Siffert, R. S.: Effect of staples on epiphyseal growth, J. Bone & Joint Surg. 38A:1077, 1956.
12. Strobino, L. J., French, G. O., and Colonna, P. C.: The effect of increasing tensions on the growth of epiphyseal bone, Surg., Gynec. & Obst. 95:694, 1952.
13. Thorndike, Augustus: Athletic Injuries, ed. 4, Philadelphia, Lea and Febiger, 1956.
14. Watson-Jones, R.: Fractures and Joint Injuries, ed. 4, London, Livingstone, 1955.

Effectos de Trauma Super le Epiphyse

Summario in Interlingua

Le sequente observationes es describite e discutite:

1. In juveniles ancora crescente le junction meta-epiphysal es plus exponite a disruption partial o complete per un effortio tangential o fortias compressori que le structuras ligamentose que supporta le articulation per se. Es recommendate urgentemente le adequate examine roentgenologic al tempore del trauma e, ancora plus urgentemente, le effectuation, durante un periodo adequate, de observationes post-tractamental de omne minor contorsiones e non-fracturate lesiones in le vicinitate del articulationes de juveniles in stadio de crescentia, in consideration del possibile occurrentia de irregularitates crescential. Il es solmente per regular examines roentgenologic post le tractamento que tal disturbationes del contorno articular pote esser demonstrate e que le appropriate mesuras pote esser instituite a un tempore quando il es ancora possibile reverter le tendentia in question.

2. Es signalate que il es possibile e practic de passar lisie filos metallic a transverso le portiones central del junctiones epi-metaphysal e de fixar assi dislocationes de fracturas del epiphyse facilemente, securmente, e—al longo—con le production de multo minus trauma que lo que resulta necessari-

mente ab repetite manipulationes fortiate e le mantenentia del dislocationes in un position extreme durante le processo curatori.

3. Es monstrate que le crescentia epiphysal pote esser relentate per dispositivos fixatori trans le junction meta-epiphysal, i.e., usualmente, crampas que effectua inter epiphyse e metaphyse un compression sufficiente pro arrestar le crescentia epiphysal. Iste arresto del crescentia es temporari usque a un certe puncto, sed illo deveni irreversibile quando osso appositional es formate a transverso le linea epiphysal. Es etiam monstrate que le arresto complete del epiphyse es effectuate per alterar le anatomia del margines meta-epiphysal de maniera a render possibile le effectuation de epiphysodese per le crescentia de osso appositional.

15

Slipping of the Upper Femoral Epiphysis

BECKETT HOWORTH, M.D., Med.Sc.D.

HISTORY

The early history of slipping of the upper, or capital, femoral epiphysis, or epiphysiolisthesis, is confused because of the variety of names applied to the disease, a lack of clear differentiation betweeen this lesion and other diseases and abnormalities of the hip, the lack of roentgenography, the paucity of observations of the pathology and the difficulties arising out of the publication of the early reports in several different languages or as obscure paragraphs in some other report. This slipping epiphysis was also called traumatic epiphyseal separation, epiphyseal fracture, adolescent rickets, bending of the neck of the femur, adolescent or epiphyseal coxa vara, arthritis deformans and femoral osteochondritis of adolescence.

The author has located and read in the original all the early references in English and French, except the cases credited to de Hilden and to Verduc, which could not be found. It appears that the earliest recognition of displacement of the capital femoral epiphysis occurs in the French literature. Paré (1572) says, "The epiphysis of the head of the femur sometimes separates in such a way that the surgeon is misled, thinking there is a dislocation instead of a separation of the epiphysis" In the middle of the 17th century de Hilden is credited by Rognetta with reporting a case of slipping epiphysis, and Verduc, about 1694, with another by Spillman. Petit, in 1709, in the chapter on fracture of the neck of the femur, discusses slipped epiphysis, the examination and the differentiation from fracture of the neck of the femur. There is a similar discussion by Duverney in 1751, although he considered the slipping to be related to disease, such as vérole (syphilis or tuberculosis) or scurvy. Sabatier, in 1768, in writing on the subject of fracture of the neck of the femur, includes the case report of a boy 15 years old, very likely a slipped epiphysis. Richerand's discussion of epiphyseal displacement in his thesis on fracture of the neck of the femur (1796) is essentially a restatement of the observations of Paré, Petit and Duverney. This is also true of Boyer's account in his *Treatise on Surgical Maladies* (1814). Dupuytren merely calls attention to epiphyseal displacement as a consequence of falls on the hip in children (1832).

Sir Astley Cooper does not mention epiphyseal slipping in his *Treatise on Dislocations and Fracture of the Joints.* Liston simply ventures the statement that it may occasionally occur. Post, in 1840, presents a case report with the correct diagnosis. Single-case reports were added by South (1847); Hutchinson (1866); Bousseau (1867); Maunder (1875); Stetter (1877); Wardner and Smith (1877); Barton (1883); Hamilton (1884); Ashurst (1884) —he believed that violence to the hip was the etiologic factor, as indeed it must have been in his case and some of the others; Smith (1886); and Monks (1886), who described bilateral slipping but ascribed it to arthritis deformans with softening of the neck. Fiorani, in 1881, reported 15 young children with a special form of limping, but

it appears that none of these was a slipped epiphysis. Colignon, in 1869, collected 10 cases from the literature.

Bousseau's (1867) was an autopsy report of a boy 15 years old with a severely traumatic displacement of the epiphysis; the boy was badly mauled by the wheel of a heavily loaded cart, and he died the next day. Wright, in 1887, recorded the autopsy of a woman 40 years old with a complete separation of the femoral head, apparently since childhood, but no history was available. Müller, in 1888, reported from Bruns' clinic 4 cases of "bending of the neck of the femur." One had been resected under the mistaken diagnosis of tuberculosis, and the pathology was described, the first true pathologic report of the disease which we are discussing.

The 1890's were years of activity and progress in the understanding of slipping of the capital femoral epiphysis. Case reports were added by Rotter in 1890, Hamilton (3) in 1891, Bradford (3), Davies-Colley, Hutchinson and Mayland in 1892, Battle (4), Whitman (4), Sturrock, Hoffa, and Hofmeister in 1894, Harte in 1897. Resection of the head of the femur as a surgical procedure was done more frequently in this decade, especially in Germany, and provided an opportunity for further gross and microscopic studies of slipping epiphysis. Davies-Colley removed a femoral head in 1892, described the gross deformity and stopped at that. In the same year Schultz performed a resection 3 years after the onset of symptoms in a girl of 14, and after careful study of the specimen concluded, as Müller had, that the lesion was a late form of rickets. Rushmore in the same year resected a femoral head, but in this case the separation was induced surgically. A boy of 16 dislocated one hip doing a split; an open reduction was done, but the dislocation recurred. A closed reduction was attempted. Subsequent open reduction revealed separation of the head from the neck, and the head was removed. Kocher, in 1896, removed two displaced femoral heads, describing and discussing the deformity. Maydl reported a resected head in 1897, Hofmeister in 1898 a traumatic displacement as well as a necrotic head following surgical infection. Sprengel in the same year described two removed heads following acute traumatic slips.

Vigorous discussion took place during this decade regarding etiology and terminology. The disease was ascribed variously to late rickets, osteochondritis, arthritis deformans, a mysterious bending of the neck of the femur of unknown origin, or acute trauma. The last-mentioned etiology was sometimes correct, as a number of the cases had had severe falls downstairs, from a height of 15 to 20 feet, or had been struck by carriages. The term *coxa vara* was suggested independently by Hofmeister and by Kocher in 1894 and for some years dominated the literature but added to the confusion by its lack of specificity. The subject of coxa vara was reviewed by de Quervain in 1898, and by Joachimsthal and by Alsberg in 1899 and 1900. Poland, in 1898, in his classic monograph *Traumatic Separation of the Epiphyses* reviewed the literature of slipping of the capital femoral epiphysis, from which he had collected 31 cases, and added a case of his own. However, it was the advent of roentgenography in 1895 which paved the way for a really great advance in the diagnosis, the understanding and the treatment of epiphysiolisthesis.

Keetley, in 1888, reported a girl 20 years old with deformity of the femur and wedging round back which he ascribed to adolescent rickets He performed a subtrochanteric wedge osteotomy of the femur for correction of the deformity; the follow-up period was 6 weeks. This deformity may have been due to an old slipped epiphysis. The femoral head resections in the 1890's have been mentioned. Sturrock, in 1894, used a pin for internal fixation of the epiphysis to the neck of the femur but had to remove it 2 days later because of infection. Bradford, in 1895, performed an osteotomy of the

femoral neck for correction of the deformity. Poland, in 1898, resected the protruding anterosuperior corner of the neck. These operations presented new opportunities for studying the deformity and the disease.

In the next two decades there were further reports of interest. Additional specimens were secured by resection of the femoral head by Rammstedt (4) in 1900, one each by Haedke in 1902, Schlesinger in 1905 and Helbing in 1906, and Frangenheim (6) in 1909. Curiously, only the bone and the cartilage were examined; no attention was paid to the soft tissues, the synovial membrane, the periosteum and the blood vessels. The clinical reports of this period included some roentgenographic findings, sometimes reproductions of the roentgenograms. The series reported were more clearly differentiated from other diseases and abnormalities of the hip. Interesting reviews were made available by Zesas (1) in 1904 and 1911, Perrin (4) in 1912, Kermisson (5) in 1897, 1912, 1913 and 1918, Drehmann (3) in 1911 and Elmslie (2) in 1907 and 1911. Hoffa reported the results of resection of the head of the femur in 4 cases and of subtrochanteric osteotomy for correction of deformity in 2 hips. Whitman, in 1909, reported 8 hips treated by open reduction of the slipping, and Wilson in 1924 presented 7 hips so treated. Additional pathologic and clinical data were accumulated, and further discussions regarding etiology appeared, but the specific cause of the lesion was not discovered.

Key, in 1926, presented his candidate's thesis for the American Orthopaedic Association, an excellent review of the literature of slipping epiphysis, with a report of 24 cases observed at the Massachusetts General Hospital in the years 1904 to 1922, fourteen of which had been followed long enough for evaluation of treatment. This remains the classic report on the subject in the American literature. Ferguson and Howorth, in 1931, reported 70 cases seen at the New York Orthopaedic Hospital in the previous 20 years, treated by various methods. The clinical data were analyzed, and a clear picture of the disease was presented: four principal stages of the disease were identified, and the roentgenographic features of the various stages were reported; its natural course was described, and the results of the various treatments were evaluated in relation to the natural course. Further, the operation of inserting bone pegs across the epiphyseal plate to produce healing of the disease and union of the epiphysis was reported. These features have been developed further by Howorth in subsequent reports, with special emphasis on the gross and the microscopic soft-tissue pathology which had not been reported previously and the results of the pegging operation in about 200 cases. In the past 25 years the literature has contained reports of additional series of cases, mostly small, treated by the methods reported earlier, and recently there have been attempts to revive open reduction as a treatment of choice, but we do not yet know the exact cause of the disease or how to prevent it.

ETIOLOGY

Slipping of the upper femoral epiphysis is one of the group of osteochondroses, which include at the hip coxa plana and coxa magna, at the knee Osgood-Schatter and Blount-Barber disease, in the foot apophysitis of the calcaneus, Köhler's disease of the navicular and Freiberg's infraction of the second metatarsal head, also epiphysitis or Scheuermann's disease of the spine, Kienböck's disease of the carpal navicular and osteochondritis dissecans of various epiphyses, as well as several minor and rare entities. All are characterized by a vascular disturbance, trauma being a variable factor, and each by a typical age distribution; only Freiberg's and Kienböck's disease occur in adults.

Epiphysiolisthesis is slightly more common in boys than in girls, whereas coxa plana is six times as common in boys. The

age distribution was 8 to 17 years, but the two youngest cases, 8 and 9, were associated with frank severe trauma. The preslipping stage begins about one year earlier than the slipping stage, especially in the younger children, the average age being 12 years. The average age for slipping was 13.5 years, about two years earlier in girls than in boys, because of the earlier skeletal development. The right hip was involved in 38 per cent of the cases, the left in 48 per cent, whereas 14 per cent were bilateral. Bilaterality is less common with early diagnosis and consequent protection of the second hip.

Sixty-five per cent of the children were significantly above average height or weight, but only half were of the endocrine type. Since half of the children were not of the endocrine type, and the disease is preponderantly unilateral, it is difficult to explain it on a purely endocrine basis Tonsillectomy had been performed within a few months of onset in half of the children, and other infections were present in 20 per cent of the cases, but no specific relationship of infection to the disease could be demonstrated, and there were no bacteriologic or pathologic findings in the hips to indicate infection. Trauma was absent in most of the cases at or near onset, and when present was usually trivial, e. g., stepping off the curb or slipping on the pavement. Although trauma does sometimes initiate a slip in a vulnerable epiphysis, in the vast majority of cases it is really an incident in the course of the disease rather than the cause. It appears that the most important factor in the etiology of the disease is rapid growth and excessive vascularization of the epiphyseal plate, with consequent softening and weakening at the junction of the neck with the capital epiphysis; the strains of weight-bearing and activity are contributory.

The typical child with slipping epiphysis is tall for his age, overweight, or both, often of the flabby type, with weak arches and knock knees, and clumsy in physical activity. Many of them walk with the legs in external rotation, and poor posture is common.

PATHOLOGY

Open operations were done at the New York Orthopaedic Hospital on nearly 200 hips with slipping epiphysis in various stages. The soft tissues, as well as the head and the neck, were studied grossly, and microscopic sections were made of the soft tissues, as well as sections from the neck across the epiphyseal plate into the head. In the preslipping stage the synovial membrane is swollen, edematous, vascular and villous. There are similar but milder changes in the capsule. Microscopic sections reveal hypervascularity with perivascular lymphocytic infiltration, wandering cells and scattered plasma cells. At the junction of the head and the neck a bluish zone is seen, and there are decalcification and hypervascularity at the junction of the neck and the epiphyseal plate. The femoral head and the acetabulum appear to be normal.

Displacement of the head is characteristically downward and backward on the neck, with a tilt into varus. Sometimes the downward displacement is predominant, at others the backward displacement. Rarely is a valgus displacement seen. Displacement varies from a minimal amount to complete displacement of the head under the neck. However, without surgical intervention, separation of the head from the neck is rare, as the slipping usually is gradual, often in stages, and the periosteum of the neck tends to keep up with the slipping. Furthermore, the inferior angle between the neck and the head tends to fill promptly with callus, so that there may be one or more zones of callus of different ages. The callus becomes mature and strong in 2 or 3 months, tending greatly to interfere with reduction but not preventing further displacement unless the softening of the epiphyseal junction heals. The slip may be recognized by a jog superiorly and anteriorly at the junction of the

head and the neck, and by the sharp angle between the head and the neck inferiorly, unless it has been smoothed out by callus. There is a wider bluish zone at the junction, often punctuated with small islands of bone projecting through it. There is a redundant mass of synovial tissue in the inferior angle. Microscopically, the synovial membrane tends in time to become less edematous and vascular, somewhat scarred and inelastic. There is separation between the neck and the epiphyseal plate, in some cases suggesting avulsion, with degenerative changes in the cartilage of the plate.

After several months the inflammatory process tends to subside and the softening at the epiphyseal junction to heal, earlier in the older children. However, healing and further slipping may overlap somewhat. The synovial membrane and capsule become more scarred and inelastic. The epiphyseal junction heals and becomes firm again, presently closes and ossifies, but this process may require from 2 to 3 years. The jog superiorly and inferiorly tends to become smoother, and the periosteum heals over the slip. Degenerative changes do not occur in the head unless the head is separated from the neck in the course of treatment, as the circulation to the head remains adequate. The residual deformity remains, and, with a moderate or a severe slip, osteoarthritic changes may be seen in middle or late adult life. These changes tend to occur earlier and to be more severe with surgical intervention. There is osteophytic production at the junction of the head and the neck, often at the superior acetabular margin. The head may become flattened superiorly, with degenerative changes and thinning of the articular cartilage.

STAGES

The disease may conveniently be divided into the preslipping, the slipping, the healing and the residual stages The preslipping stage is characterized by mild symptoms and signs, due to synovitis, with early pathologic and roentgenographic changes. The slipping stage is marked by similar but more pronounced changes, with the clinical, the pathologic and the roentgenographic evidences of slipping. In the healing stage the inflammatory symptoms and signs subside, and the epiphyseal junction heals and closes. The residual stage is marked by the evidences of deformity and later may be associated with osteoarthritis.

SYMPTOMS

The early symptoms are those of mild synovitis: slight pain, limp, stiffness and disability. The pain is worse with activity, relieved by rest. It is located in the groin, front or inner side of the thigh, or inner side of the knee. This radiation of the pain sometimes causes confusion, especially when examination is limited to the knee and the thigh, and thus the diagnosis may be missed. When slipping occurs, the symptoms tend to become more evident; if the slipping is sudden or marked, the pain may be severe, and the child may be unable to put weight on the leg. As the disease heals the symptoms tend to subside. However, with successive slippings there may be exacerbations. The residual symptoms are those of deformity, varying in degree, such as limp due to shortening and external rotation of the leg. Later the pain, stiffness and disability of osteoarthritis may supervene.

SIGNS

The early signs of the preslipping stage are a slight limp and limitation of abduction and internal rotation with slight pain and spasm at the extremes. Slipping results in further limitation of motion, increase in pain and spasm, shortening due to displacement, and the typical limp due to pain, shortening and external rotation deformity. Flexion of the hip is associated with external rotation and abduction, whereas straight flexion is not possible. The femoral head can be felt in the buttock, especially on rotation These two signs are pathognomonic. As healing

proceeds, the acute signs subside, but some limitation of motion, shortening and limp persist, due to the mechanics of the deformity. Later, osteoarthritis develops and is associated with increasing pain, limp and restriction of motion.

LABORATORY TESTS

The blood count, blood calcium, phosphorus and phosphatase and other common laboratory tests have been consistently normal. The erythrocyte sedimentation rate usually is elevated slightly, sometimes moderately. Cultures of the synovia, synovial membrane, cartilage and bone from the hip have uniformly yielded no growth.

ROENTGENOGRAMS

Roentgenograms are most helpful in confirming the diagnosis of slipping of the epiphysis and in following its course and the effects of treatment. They are particularly valuable for the early diagnosis of the disease, when it is important to include roentgenograms of the opposite hip for comparison. Anteroposterior and lateral views should be obtained; the latter can best be obtained with the hip flexed to 90° and abducted about 40°, with the patient, the film and the roentgen tube in the same position as for the anteroposterior view. However, when there is much pain and spasm or limitation of motion, the position must be modified accordingly.

The early roentgenographic changes are a globular swelling of the hip-joint capsule and decalcification, widening and irregularity on the neck side of the epiphyseal line, without displacement of the epiphysis. Slipping is downward or backward, or both. Downward slipping is evident in the anteroposterior view, whereas posterior displacement often is recognized more easily in the lateral view; in doubtful cases the latter view, with the normal hip for comparison, often is more helpful. The changes in the curves of the head and the neck in the anteroposterior view, flattening superiorly and abruptness inferiorly, and in the lateral view a jog anteriorly with overriding posteriorly are distinctive. The tilt of the head posteriorly and into varus is also recognizable. Soon, however, the prominent superior corner of the neck begins to smooth off by absorption and the abrupt inferior angle to fill in with callus, so that the displacement is not as conspicuous, *but the distortion of the basic curves* persists.

In the healing stage the swelling of the capsule subsides, the epiphyseal junction recalcifies and heals, and in time the epiphyseal line is obliterated, only the deformity remaining. *If there has been damage to the* blood supply of the epiphysis in the course of treatment, the joint space may become thinner, and the degenerative changes characteristic of aseptic necrosis in various degrees appear in the head, with marginal lipping. These changes may be obvious in a few months, or they may be delayed for a year or more. The residual stage is characterized by deformity, sometimes also by the quiescent phases of aspetic necrosis; eventually some degree of osteoarthritis with thinning of the joint space, marginal lipping of the head and acetabulum, condensation of bone at the articular surfaces and, sometimes, cystic degeneration may supervene. Slipped epiphysis is probably the most common cause of osteoarthritis of the hip, often in the past called by the nonspecific term *malum coxae senilis.*

DIFFERENTIAL DIAGNOSIS

Any child between the ages of 8 and 17 years with a limp, pain in the thigh and, on careful examination, limitation of motion at the hip with pain and spasm at the extremes should be suspected of having a slipping epiphysis; he should immediately be taken completely off weight-bearing, and roentgenograms should be made of the hip. If there is doubt, the child should be kept off weight-bearing, for several weeks if necessary, until symptoms and signs have subsided, until serial roentgenograms can be

had and until the diagnosis can be established or disproven. Slipping epiphysis is the most common disease of the hip in children of this age group, but the diagnosis often is missed, at least until there has been a serious slip. It is most important that the *diagnosis be made early, for the results of* proper treatment in the early stage are excellent, but it is difficult to ensure a good result after moderate or severe slipping has occurred. The features of the other diseases that affect the hips of children of this age group, such as tuberculosis and other infections, are so characteristically different and so well known that differential diagnosis is rarely a problem. True fracture of the neck of the femur is rare in children of these ages. *The most important aid in diagnosis* is to think of the possibility of slipping epiphysis, examine the hip carefully and obtain good roentgenograms.

EVALUATION OF TREATMENT

The essentials for proper evaluation of the results of treatment are a knowledge of the natural course of the disease and careful, accurate appraisal of the hip with the normal hip as a standard. It should be recognized that the disease always heals, regardless of treatment, and that the results of untreated hips often are good, sometimes better than some of the results of treatment Treatment is of no value unless the result is better than if the hip were not treated. Further, it should be recognized that aseptic necrosis does not occur in the untreated hip and that untreated hips rarely cause serious trouble before middle or late adult life. An *excellent result is a child with a hip which,* with a careful and an honest appraisal, has no limp, pain or disability for walking, running or sports, no shortening, full and free



FIG. 1. A 14-year-old boy. A moderate slip with no treatment and a good result *(Left)* July, 1946. A moderate slip downward and backward. The epiphyseal line is wide, with irregular decalcification on the distal side. There is mature callus in the inferior angle. *(Right)* September, 1949, 3 years later. The lesion healed nicely without treatment and without degenerative changes in the head The patient had no pain and no disability; limp was negligible. Abduction and internal rotation were slightly limited Osteoarthritis may develop eventually because of incongruity of the head-neck junction with the superior acetabular margin, but the symptoms will be slight, at least until middle life. The effects of treatment should be evaluated in relation to the results of untreated hips and in terms of the normal hip. (Howorth, B.: J. Internat. Coll. Surgeons 20:717)

motion and no deformity. After the untreated hip heals symptoms and signs will be only those of the residual deformity, and if the slip is less than ½ inch they will be slight and not disabling. It is difficult to secure a better result by treatment.

The prime purpose of treatment should be to prevent significant slipping of the epiphysis. If this is accomplished, no other treatment will be necessary, and the result will be excellent.

TREATMENT BY PROTECTION OF THE HIP

Since the disease always heals, it may be treated by bed rest. However, many months may be required for healing, and there is some risk that the epiphysis will slip even in bed, especially if the child is vigorous and active. Normally this epiphyseal plate closes at about 16 years of age in girls, 18 in boys. The younger the child, the longer the interval for healing. Additional protection may be secured with a plaster cast on the hip, but there is a marked tendency for this to produce permanent limitation of motion, so that this method is to be condemned. Wright and King have just reported a 10-year experience with 35 hips employing short leg casts with cross bars to maintain internal rotation without attempting reduction of displacement, with satisfactory results in a large percentage of the cases. Kite reports a favorable experience with 9 hips treated similarly, except that an attempt was made to reduce acute slips by gentle manipulation. Certainly methods of this sort are to be preferred to vigorous manipulation, open reduction or complete immobilization.

Some protection may be obtained by means of traction on the affected leg; this method is to be recommended for a week or two when there is much pain and spasm, but prolonged traction is not advisable. After



FIGS. 2 and 3. A 13-year-old girl. Partial closed reduction of a moderate slip, immobilized in a cast. The result was fair, as usual with this type of treatment. (Howorth, B.: J. Internat. Coll. Surgeons 20:718)

FIG. 2 *(Above). (Left)* September 24, 1930. Symptoms for 3 months; index of motion, 95. Moderate posterior displacement as indicated by the shallow head and the elliptical epiphyseal line. Treated with bed rest for 6 months, after which weight-bearing was begun. The epiphyseal line appears to be healed. *(Right)* November 14, 1930. Three weeks after weight-bearing there was a pronounced further slip, as indicated by the new position of the head, as well as the zones of callus. Healing with bed rest alone may require many months. Operation November, 1930. A closed reduction was attempted, with partial reduction of the downward dislocation. A double plaster spica was worn for 3 months.



FIG. 3. *(Left)* March 5, 1931. Four months after reduction. The epiphyseal plate has healed and is closing There are mild degenerative changes in the head, and the joint space is slightly thin. *(Center)* November, 1932. Two years after reduction. The epiphyseal plate is closed and obliterated. There is moderate decalcification at the hip, also slight irregular ossification, and the joint space is thin superiorly. Pain, limp and disability are present. The index of motion is 50. *(Right)* October, 1945. Fifteen years after reduction. The contour of the head is irregular, with moderate marginal lipping. Ossification is slightly irregular, and the joint space is thin superiorly. Pain, limp and disability have increased The index of motion is 40.

pain and spasm have subsided, the child may be ambulant with crutches, with or without a leg sling of the Sam Browne belt type, without weight-bearing on the affected side, but it must be realized that children are not always dependable and that a large clumsy child usually is in danger of falling, with consequent slipping of the epiphysis. A truly non-weight-bearing brace may be employed, with a built-up shoe on the normal side, but again there is some risk of injury to the hip, and extension is not the most favorable position for the compromised circulation of the hip joint. Such methods of treatment may well be combined with pool exercises, if safety can be ensured. The major disadvantage of such methods is the long time required, with the consequent waste of time for other purposes, extra expense and the many psychological, as well as physical, disadvantages of converting the child into a long-term invalid.

CLOSED REDUCTION BY MANIPULATION

Whitman, in 1894, advocated reduction of the displacement by manipulation, abduction and internal rotation, followed by fixation in a plaster spica. This method of reduction is successful only if there is a single very recent slip, with no callus in the inferior angle. The manipulation should be gentle, not forceful. Immobilization in plaster, or even in prolonged traction, is likely to result in a partially stiff hip. Accordingly, internal fixation, as recommended by Wilson with the Smith-Petersen nail, or with small nails, wires or screws, is preferable, as motion is permissible and stiffness is less likely. Unfortunately, only a small percentage of the hips with moderate or more slipping are of this easily reducible type. We have shown that those with callus in the inferior angle or with partial union of the epiphysis are not reduced by this method; only two of seventeen hips were anatomically improved. One may be deceived by the changed position of the hip when the roentgenogram is made after manipulation. Aseptic necrosis followed the manipulation in several hips.

REDUCTION BY TRACTION

The single recent slip, without callus, may be reduced by manual or skeletal leg traction and immobilized by internal fixation. However, in most cases very strong



FIGS. 4 and 5. A 13-year-old boy. Closed reduction and fixation with Smith-Petersen nail. A good clinical and anatomic result, usual for this type of treatment with a fresh slip and no callus. No degenerative changes due to treatment. (Howorth, B.: J. Internat. Coll. Surgeons 20:720)

FIG. 4 *(Above). (Left)* December 27, 1940. Symptoms, 3 weeks; index of motion, 45. Head displaced downward ½ inch, posteriorly ¾ inch. The epiphyseal line is wide, with irregular decalcification distally. The joint space is normal. Operation January 8, 1941. Closed reduction with traction, internal rotation and abduction. Smith-Petersen nail inserted. *(Center)* January 17, 1941. Nine days afer reduction. A fairly good reduction, but slight downward and moderate posterior displacement remain, due to the old callus. The nail is well placed; the epiphyseal line is healing. *(Right)* June 18, 1941. Five months after reduction. The epiphyseal line has healed and closed. The head is healthy.

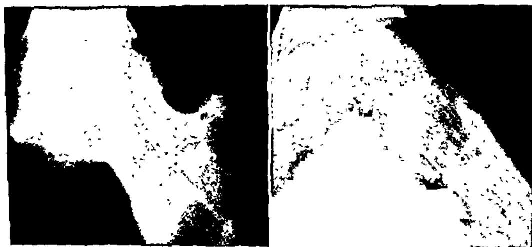


FIG. 5. April, 1945. Four and a half years after reduction. The nail has been removed. The epiphyseal line is obliterated, the head is healthy and the joint space is normal. There are slight residual deformity and moderate lipping at the posterior margin. No pain or limp; index of motion, 95. The patient is working as a messenger without handicap.

traction is required to overcome the resistance of the callus inferiorly. The author employed this method in seven cases, obtaining reduction of the downward but not of the posterior displacement. The hips were immobilized for from 10 to 12 weeks. The final range of motion was good in only one hip, poor in seven; aseptic necrosis occurred in six of the hips, due to circulatory damage. This is certainly not a good method. The

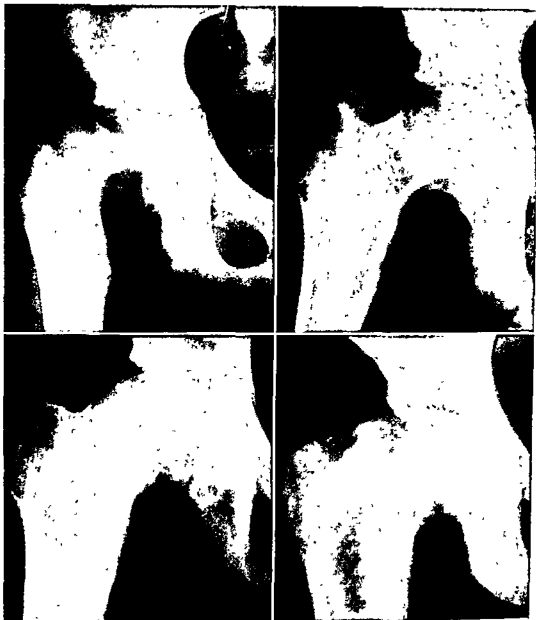


FIG. 6. A 13-year-old boy. Reduction by strong traction with plaster immobilization. A fair clinical and anatomic result, one of the better results of this type of treatment. However, degenerative changes developed in the head, and the result became much worse.

(Top, left) March 9, 1931. Symptoms, 6 months; index of motion, 22. Head displaced downward and posteriorly ¾ inch. There are two zones of callus indicating an old slip as well as a fresh one. The joint space is normal. Operation, March 18, 1931. Closed reduction by strong traction, with immobilization in a double plaster spica for 3 months *(Top, right)* June 23, 1931. Three months after reduction. A fair reduction with slight downward and moderate posterior displacement The epiphyseal line has healed and is closing. Slight decalcification The joint space is thin. *(Bottom, left)* November 18, 1933. Two and a half years after reduction. The epiphyseal line is closed and obliterated. The head is healthy; the deformity is unchanged. The index of motion is 34. *(Bottom, right)* April, 1945. Fourteen years after reduction. There are moderate cystic changes in the head and the acetabular roof. The joint space is thin superiorly The index of motion is 20. Moderate pain and disability. (Howorth, B : J. Internat. Coll. Surgeons 20:719)

results might have been better with internal fixation and early motion, but it is probable that the traumatism of the reduction was the major factor in the poor results.

OPEN REDUCTION

Open reduction of the displaced epiphysis was advocated by Whitman in 1909 and by Wilson in 1924. Open reduction was done for 31 hips at the New York Orthopaedic Hospital in the period 1920 to 1946. The head was freed and replaced on the neck in 25 hips, and the deformity was corrected by cuneiform osteotomy of the adjacent neck in six. The anatomic results were good in nearly all the hips. Twenty were immobilized in casts for from 10 to 12 weeks; good clinical results were obtained in

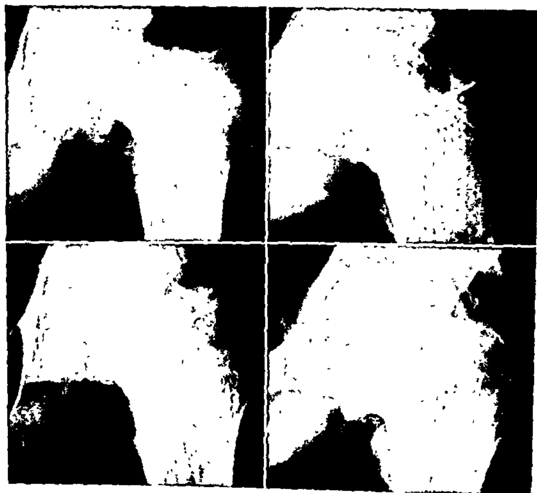


FIG. 7. A 17-year-old boy. Open reduction with plaster immobilization. Head separated from neck at operation. A fair clinical and anatomic result, average for this type of treatment. *(Top, left)* October 3, 1932. Symptoms, 6 months; index of motion, 48. Head displaced downward ¾ inch, posteriorly ½ inch. Shaft in external rotation Epiphyseal line wide, irregular and decalcified, but healing. Mature callus in inferior angle. Joint space normal. Operation October 31, 1932. Open reduction. Double plaster spica 3 months. *(Top, right)* April 22, 1933. Six months after operation. Head well reduced into slight valgus. Epiphyseal line healed and closed. Slight decalcification of head. Joint space thin. *(Bottom, left)* December 27, 1933. Fourteen months after operation. Epiphyseal line obliterated. Head flat laterally, with irregular decalcification of lateral fourth. Joint space very thin. *(Bottom, right)* September 24, 1938. Six years after operation. Head bell shaped, well ossified. Joint space thin. Lipping at margins of head. Neck short, shaft in external rotation. Slight pain and limp, but active. Index of motion, 40. Compare with Figure 1. (Howorth, B.: J. Internat. Coll. Surgeons 20:721)



FIG. 8. A 14-year-old boy. *Open reduction; head separated from neck; fixation with nail* A poor clinical and roentgenographic result because of damage to circulation of head. *(Left)* February 20, 1940. *Symptoms, 4 months. Head displaced downward 1* inch, posteriorly 1 inch. Mature callus inferiorly. Epiphyseal line healed, probably closed. Joint space normal. Operation February 28, 1940. Open reduction, fixation with nail. *(Center)* April 5, 1940. Six weeks after operation. Well reduced in slight valgus, but slight downward and posterior displacement. Epiphyseal line healing and closing. Slight decalcification. *(Right)* September 27, 1940. *Seven months after operation.* Head flat superiorly, cortex irregular, irregular decalcification Joint space very thin Pain, limp, disability. Index of motion, 20. A hip fusion operation was done in October, 1941. (Howorth, B.: J. Internat. Coll. Surgeons 20:722)

only two of these; marked limitation of motion and aseptic necrosis were the rule. Internal fixation with a Smith-Petersen nail was obtained in seven hips, with a small nail in two, and with a Lippmann screw in two. The range of motion was better in this group than in those immobilized, but degenerative changes occurred in four of the hips, and arthritic lipping was common in *those followed for several years.* The operations were done with gentleness and care by orthopaedists skilled in hip surgery, to avoid damage to the blood supply of the hip. In recent years several attempts have been made to revive or justify open reduction, but aseptic necrosis and arthritic lipping have been seen in a fair portion of the cases presented, and some of them had poor reductions or so little slipping that reduction was not justified. The obvious cause of the degenerative changes is the damage to the blood supply at the time of surgery. The risk of nailing is particularly great if the nail is driven too far and must be withdrawn, or if it is driven in the wrong direction and the nailing must be repeated. The author has discussed open reduction with many orthopaedists of this and other countries, and it is generally agreed that the incidence of aseptic necrosis after open reduction is too great to justify its use Certainly, the operation should not be used generally and, if at all, only in the most favorable circumstances and by the most experienced and skilled orthopaedists.

PARTIAL OSTECTOMY OF THE FEMORAL NECK

Poland in 1898, Whitman in 1909 and Vulpius and Stoffel in 1913 reported one *case each in which, without attempting to* reduce the displacement, the projecting anterosuperior margin of the neck was trimmed away. Heyman, Herndon and Strong have just reported favorable results with this operation for 21 hips; in the early cases a bone graft also was used.

SUBTROCHANTERIC OSTEOTOMY

External rotation deformity may be corrected, abduction improved and the femoral head brought into better relationship with the acetabulum by means of subtrochanteric



FIG. 9. A 17-year-old boy. Open reduction; head separated from neck; fixation with Smith-Petersen nail, which entered acetabulum. A poor clinical and roentgenographic result, with early degenerative changes. *(Top, left)* December 2, 1940. Symptoms, 9 months; index of motion, 15. Head displaced downward ¾ inch, posteriorly ¾ inch. Mature callus in inferior angle; superior angle of neck rounded Epiphyseal line wide and irregular, but healing and partially united. Operation December 2, 1940. Open reduction; fixation with Smith-Petersen nail *(Top, right)* December 9, 1940. Seven days after operation Head in valgus but displaced posteriorly; shaft rotated externally. Nail well centered but enters acetabulum. *(Bottom, left)* April 4, 1941. Four months after operation Nail removed Head flat superiorly, with area of degeneration and decalcification suggestive of osteochondritis dissecans. Epiphyseal line healed and closed. Joint space very thin. *(Bottom, right) March 19, 1948.* Seven years after operation. Head wedge shaped. Epiphyseal line obliterated. Joint space very thin. Slight lipping of acetabular margin. (Howorth, B.: J. Internat Coll. Surgeons 20:723)



FIG. 10. A 13-year-old boy. Open reduction; head separated from neck; fixation with Lippman screw. A fair clinical and roentgenographic result, with early osteoarthritis. *(Left)* August 14, 1936. Acute symptoms, 4 days; index of motion, 21. Head displaced downward ¾ inch, posteriorly ¼ inch. Epiphyseal line wide and irregular. Mature callus fills part of inferior angle and appears to be a part of the neck, but there has been a further very recent slip. Joint space normal. Operation August 21, 1936. Open reduction; fixation with Lippman screw. *(Center)* October 3, 1936. Six weeks after operation. Head very well reduced. Screw well centered and secure. Very slight decalcification. Joint space normal. Screw removed 2 days later. *(Right)* September 20, 1939. Three years after operation. Head solidly united; epiphyseal line obliterated. Head irregularly ossified and bell shaped with marginal lipping. Head subluxated laterally. (Howorth, B.: J. Internat. Coll Surgeons 20:724)

osteotomy. *The risk of aseptic necrosis following subtrochanteric osteotomy is minimal.* If the epiphyseal displacement is slight, *or even moderate, there usually will be enough improvement with healing of the lesion so that osteotomy will not be required.* With marked displacement osteotomy usually will be necessary, most of all for swinging the jog at the head-neck junction superiorly away from the acetabular margin. It is preferable to defer the osteotomy until pain and spasm have subsided and the lesion has healed.

Subtrochanteric osteotomy was done in a small series of cases with a long leg hip spica including the opposite thigh for immobilization. It was found that immobilization for 12 weeks tended to result in permanent limitation of motion and that control of the relationship of the fragments in the cast was difficult or impossible. With the hip in extension the pull of the iliopsoas muscle resulted in forward angulation at the osteotomy site, thrusting the femoral head further inferiorly in the acetabulum, shortening the femur and tending to increase extension at the expense of flexion of the hip. Possibly this outcome could be prevented by placing the hip in the cast in moderate flexion, but other difficulties would be encountered. Subsequently, internal fixation of the fragments, employing various devices, was employed with better control of the fragments and much less tendency to residual limitation of motion. However, with a movable hip, adequate and accurate fixation of the fragments technically is not easy and should be undertaken only by orthopaedists experienced in such methods. Although subtrochanteric osteotomy is an indirect method of correcting the deformity, *in most situations it is the safest method and, therefore, to be preferred.*

INTERNAL FIXATION WITHOUT REDUCTION

Fixation of the capital epiphysis to the femoral neck and prevention of slipping may

be secured with a Smith-Petersen nail, as advocated by Wilson and others, or by one or more pins, small nails or screws. Excellent results have been obtained with such methods in a large percentage of cases in a number of series. Such methods are comparatively simple and reasonably safe in the hands of orthopaedists experienced in hip

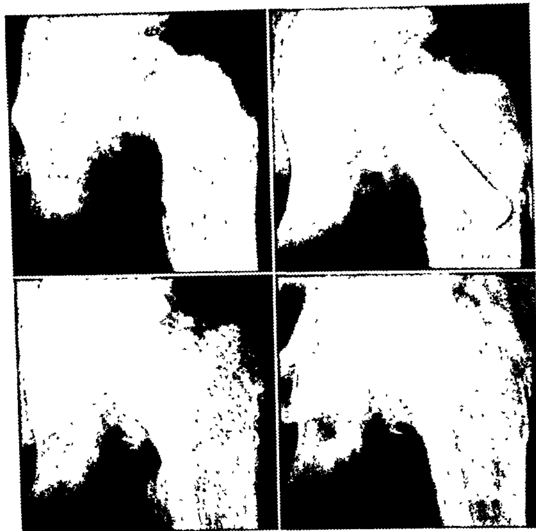


FIG. 11. A 14-year-old boy. Wedge osteotomy of neck; fixation with Smith-Petersen nail The circulation was preserved posteriorly. A fairly good initial anatomic and clinical result, followed by aseptic necrosis and a poor clinical result, not uncommon with this type of treatment. *(Top, left)* April 7, 1949. Symptoms, 6 months; index of motion, 50. Head displaced downward and posteriorly ⅝ inch. Epiphyseal line wide, with irregular decalcification. Zones of callus indicate an older and a fresh slip. Joint space normal Operation April 8, 1949. Wedge osteotomy of neck; posterior attachment and circulation preserved. Fixation with Smith-Petersen nail. Motion begun promptly. *(Top, right)* June 24, 1949. Ten weeks after operation. A moderately good reduction. One corner of the nail enters the articular cartilage of the head. Epiphyseal line has healed and is closing. The head appears to be healthy. *(Bottom, left)* September 24, 1949. Six months after operation. The epiphyseal line is closed. There are degenerative changes in the head, especially at its junction with the neck superiorly The joint space is thin. The index of motion is 40. There are pain, limp and moderate disability. *(Bottom, right)* October 9, 1950. Eighteen months after operation. The epiphyseal line is obliterated The degenerative changes are healing, with moderate residual deformity. The index of motion is 30 Pain, limp and disability have increased. (Howorth, B.: J. Internat Coll. Surgeons 20:725)



FIG. 12. A 15-year-old boy. Subtrochanteric osteotomy, with plaster immobilization. The coxa vara was improved, abduction and internal rotation were increased, but a new deformity occurred at the osteotomy site (*Left*) November 6, 1926. Symptoms, 2 years; index of motion, 68. The head is displaced downward and posteriorly ½ inch, with mature callus in the inferior angle. The epiphyseal line is wide and irregular but healing. The abrupt *superior corner of the neck has absorbed, as usual. The head* appears to be healthy. Operation March 10, 1927. Subtrochanteric osteotomy; immobilization in double plaster spica, with lower fragment abducted and rotated internally. (*Center*) March 12, 1927. Showing position of fragments in cast. (*Right*) May, 1930 Three years after osteotomy. The upper fragment has flexed, but there is solid union with reduction of the varus and increase in abduction and internal rotation. The epiphyseal line is closed. The head is healthy, and the joint space is normal, there is slight marginal lipping. (Howorth, B.: J. Internat. Coll Surgeons 20:726)



FIG. 13. Model of pegging operation, showing fenestration at junction of head and neck, through which three drill holes are made with a Nicola gouge, and bone *pegs from the iliac wing are inserted.* There should be a minimum of dissection during the exposure so as to avoid damage to the circulation of the head (Howorth, B. Textbook of Orthopaedics, Philadelphia, Saunders)

nailing. However, the possibility of damage to the circulation by such a procedure, particularly if a large flanged nail is driven in incorrectly or more than once, must be recognized. Occasionally the head has been knocked loose from the neck in driving the nail. Such complications, with resultant aseptic necrosis and tragic results, have occurred in a disturbing percentage of reported cases. Good roentgenographic control during the operation and great care should result in virtual elimination of this complication. At least two subtrochanteric fractures of the femur through the site of nail entry have been reported. Small nails or pins are



FIG. 14. A 12-year-old boy. Pegging operation, slight slip. An excellent clinical and roentgenographic result, with no degenerative changes. The usual result with this operation. *(Top, left & center)* July 25, 1946. Symptoms, 7 weeks; index of motion, 80. Slight posterior displacement. Epiphyseal line wide and irregular, with decalcification distally. Operation August 26, 1946. Bone pegging *(Top, right, & bottom, left)* October 21, 1946. Two months after operation Epiphyseal line healed and closed; head healthy; pegs absorbing. *(Bottom, right)* February 25, 1947. Six months after operation. Epiphyseal line healed and closed; head healthy; pegs being absorbed. (Howorth, B., J Internat. Coll Surgeons 20:728)

safer than the larger flanged nails. Ordinarily, this is the operation of choice when there is a slight slip (less than ½ inch) or in the preslipping stage. When the disease is active clinically and roentgenographically, it is preferable that weight-bearing be avoided until pain and spasm have subsided and the epiphyseal line has healed. This usually requires from 3 to 6 months, longer with younger children or more active disease Motion of the hip is desirable during this convalescent period, and supervised protected pool exercises are advantageous.

BONE PEGGING WITHOUT REDUCTION

It has been found in a series of more than 200 cases in the past 25 years, followed for from 1 to 17 years with an average follow-up of 7 years, that inserting bone pegs across the epiphyseal plate results in prompt healing of the disease, prevents further slipping and is not followed by aseptic necrosis. The



FIG. 17. A 16-year-old boy. Bone pegging operation; no immobilization A very good roentgenographic and clinical result despite slight slipping. *(Top, left & center)* February 21, 1947. Symptoms, 7 months. Index of motion, 79. Head displaced downward ¼ inch, posteriorly ⅜ inch. Epiphyseal line wide and irregular. Mature callus in posterior and inferior angle. Head healthy, joint space normal. Operation March 11, 1947 Three bone pegs placed across epiphyseal plate, no immobilization. Hip motions begun second day. *(Top, right & bottom, left)* May 10, 1947 Two months after pegging operation. Epiphyseal line healed and closed No further displacement. Head healthy, joint space normal. *(Bottom, center & right)* May 22, 1948. Fourteen months after pegging operation. Epiphyseal line obliterated Head healthy, joint space normal. Slight deformity due to original displacement Index of motion, 90 No pain, limp or disability Fully active, including sports. (Howorth, B.: J. Internat. Coll. Surgeons 20:730)

disability was unusual, never severe. There was slight marginal lipping in some of the hips, but no degenerative changes or aseptic necrosis in any hip, even with the strictest standards. This is in marked contrast with the epiphyses which were reduced, whatever the method The results for this entire group were better than for any reported group having internal fixation with nailing. Heyman (1949) reported good results with a similar operation for 19 hips Bone pegging of itself promotes healing of the lesion and closure of the epiphyseal plate, and thus is more physiologic. Therefore, if the surgeon is equally facile with internal fixation and bone pegging of the epiphysis, the latter is the procedure of choice.

SUMMARY

The clinical features and the roentgenographic findings, the gross and the microscopic pathology of the soft tissues, as well

as the bone and the cartilage, and the natural course of slipping of the upper femoral epiphysis have been well known for the past 25 years. The exact cause of the vascular disturbance and the softening at the epiphyseal plate which precede the slipping is not yet definitely known. It has been demonstrated repeatedly in significant numbers of



FIG. 18. A 10-year-old girl. Pegging operation failed because pegs did not cross epiphyseal plate; operation repeated successfully, with prompt healing and a good clinical and roentgenographic result. No degenerative changes. *(Top, left)* August 11, 1938. Symptoms, 3 weeks. Index of motion, 90 Epiphyseal line wide and irregular, with decalcification distally. No displacement. Operation 1: August 15, 1938. Pegging of epiphysis. *(Top, right)* April 13, 1939. Eight months after pegging. No evidence of healing Operation 2: May 15, 1939. Pegging of epiphysis. *(Bottom, left)* August 7, 1939. Three months after second pegging. Epiphyseal line healing and closing Head healthy. *(Bottom, right)* December 7, 1940. Eighteen months after second pegging Epiphyseal line obliterated, head healthy, joint space normal. No pain, limp or disability. Motions normal. (Howorth, B.: J. Internat. Coll Surgeons 20:731)

hips that separation of the epiphysis from the neck by surgical means, whether manipulation, strong traction or open operation, is followed by aseptic necrosis of the epiphysis with pain, limitation of motion and disability in too large a percentage of hips to justify such procedures except, possibly, in very unusual circumstances. It has also been demonstrated that the results of the natural healing of the disease often are better than the results following reduction of the slipping and that aseptic necrosis does not occur without surgical intervention. Further, immobilization of the slipping epiphysis, especially in extension, internal *rotation and abduction*, causes permanent limitation of motion.

Slipping of the epiphysis may be prevented sometimes by protection of the hip without immobilization, more surely by *internal fixation with nails, pins or screws*, or by the insertion of bone pegs across the epiphyseal plate. Internal fixation carries a small risk of aseptic necrosis with its consequent disability, whereas this complication has not occurred with a large series of hips treated by the bone-pegging operation. Best results are obtained when the disease is recognized early and slipping is prevented.

Early recognition of slipping epiphysis depends upon the education of the physician who first sees the child after the disease begins. Weight-bearing should be stopped as soon as the disease is suspected. Discovery of the exact cause of the disease and of a method of preventing or at least healing it promptly without surgery will be the major contributions of the future.

BIBLIOGRAPHY

Alsberg, A. Anatomische u. klinische Betrachtungen über Coxa Vara, Ztschr. orthop. Chir. 6:106-173, 1899.

———. Zur Theorie u. Differentialdiagnose der Coxa Vara, Ztschr. orthop. Chir. 7:364-375, 1900

Ashurst, J : Separation of the upper epiphysis of the femur (in fractures of the femur), Internat. Encycl Surg 4:198, 1884.

Badgley, C. E., Isaacson, A. S., Wolgamot, J. C., and Miller, J. W.: Operative therapy for slipped upper femoral epiphysis; an end result study, J. Bone & Joint Surg. 30A:19-28, 1948.

Balensweig, Irwin: Femoral osteochondritis of adolescents and sequelae, Surg., Gynec. & Obst. 43:604-614, 1926.

Barton, J. M.: Separation of the epiphysis of the head of the femur, M. News (Philadelphia) 43:43, 1883.

Battle, W. H : Traumatic separation of the upper end of the femur in early life, St Thomas's Rep. (New Series) 22:19-22, 1894.

Bousseau, M.: Disjonction épiphysaire traumatique de la tête du fémur, Bull. Soc. anatomique de Paris 42:283-286, 1867.

Boyd, H. B., Ingram, A. J., and Bourhard, H O.: The treatment of slipped upper femoral epiphysis, South. M. J. 42:551-560, 1949.

Boyer: Traité des maladies chirurgicales, vol. 3, chap. 13, p 313, Paris, 1814.

Bradford, E. H.: Three cases of separation of the epiphysis at the head of the femur, Boston M. & Surg. J. 126:212, 1892.

———: Separation of the epiphysis of the head of the femur, M. & Surg. Rep., Children's Hospital, p. 292, 1895.

Cleveland, M., Bosworth, D. M., Daly, J. N., and Hess, W. E.: Study of displaced capital femoral epiphysis, J. Bone & Joint Surg. 33A: 955-967, 1951.

Colignon, Jules: Thèse de la disjonction traumatique des épiphyses, Paris thesis, 1869.

Davies-Colley, N.: Repaired traumatic separation of the epiphysis of the head of the femur, Tr. Clin. Soc. 25:288-289, 1892.

de Quervain, F.: *De la coxa vara, Semaine méd.* 18:41-48, 1898.

Drehmann, G.: Die Coxa Vara, Ergebn. Chir. u. Orth. 2:452-487, 1911.

Dupuytren: Leçon sur les fractures du col du fémur, leçons orales de Clinique chirurg. 2:90 (Paris), 1832.

Duverney: Traité des maladies des os (trans. by S. S. Ingham, 1762), vol. 1, pp 354-375, Paris, 1751.

Elmslie, R. C.: Erasmus Wilson Lecture *on* Injury and deformity of the epiphysis of the head of the femur, coxa vara, Lancet 1:410-417, 1907.

Ferguson, A., and Howorth, B.: Slipping of the upper femoral epiphysis, J.A.M.A. 97:1867-1872, 1931.

Fiorani. Sopra una forma speciale di zoppicamento, Gazz. d. Osp. 2:717-749, 1881.

Frangenheim: Zur Aetiologie u. path. Anat. der Coxa Vara, Beitr. klin. Chir. 65:19, 1909.
———: Zur Pathologie der Osteoarthritis Deformans Juvenilis, Beitr. klin. Chir. 72:239, 1911.
Ghormley, Ralph K., and Fairchild, R. D.: Diagnosis and treatment of slipped epiphysis, J.A.M.A. 114:229-235, 1940.
Green, W. T.: Slipping of the upper femoral epiphysis, diagnostic and therapeutic considerations, Arch Surg. 50:19-32, 1945.
Haedke, M.: Zur Aetiologie der Coxa Vara, Deutsche Ztschr. Chir. 66:89-97, 1902.
Hamilton, F. H.: Fractures and Dislocations, ed. 7, chap. 29, p. 424, Philadelphia, Henry C. Lea & Son, 1884.
Hamilton, F. H., and Smith, S.: Fractures and Dislocations, p. 369, Philadelphia, Henry C. Lea & Son, 1891.
Harte, R. E.: Epiphyseal separation of the head of the femur with report of a case, Tr. Am. S. A. 15:215, 1897.
Helbing, C.: Die Coxa Vara, Ztschr. orth. Chir. 15:502-632, 1906.
Heyman, C. H., and Herndon, C. H.: Epiphyseodesis for early slipping of upper femoral epiphysis, J. Bone & Joint Surg. 36A: 539-555, 1954.
Hoffa, A.: Über Schenkelhalsbrüche in kindlichen u. jugendlichen Älter, Ztschr. orth. Chir. 11:529, 1903.
Hofmeister, F.: Zur Pathologie und Therapie der Coxa Vara, Beitr. klin. Chir. 21:299-365, 1898.
———: Coxa Vara. Eine typische Form der Schenkelhalsverbiegung, Beitr. klin. Chir. 12: 245-317, 1894. Also Joachimsthal's Handbuch der orth. Chir., pp. 362-428, Jena, 1905.
Howorth, M. B.: Slipping of the upper femoral epiphysis, Surg., Gynec. & Obst. 73:723-732, 1941.
———: Slipping of the upper femoral epiphysis, J. Bone & Joint Surg. 31A:734-747, 1949.
———: Slipping of the upper femoral epiphysis, Am. Acad. Orthop. Surgeons, Instructional Course Lectures 8:306-317, 1951.
——— Slipping of the upper femoral epiphysis, J. Internat Coll Surgeons 20:716-735, 1953
Hutchinson. Separations of the upper epiphysis of the femur, M. Times & Gazette 1:195, 1866.
——— On detachment of the epiphyseal head of the femur, Arch. Surg. 3:289, 1892.
Joachimsthal, G.: Über Wesen und Behandlung der Coxa Vara, Sammlung klin. Vortr. N. F. 215 (Chir. No. 62), pp. 1043-1057, 1899.
Keetley, C. B.: A case of rachitis adolescentium, Illus. M. News 1:7-8, 1888.
Kermisson: Difformités acquises de l'appareil locomoteur, p. 379, Paris, 1902.
Key, J. Albert: Epiphyseal coxa vara or displacement of the capital epiphysis of the femur in adolescence, J. Bone & Joint Surg. 8:53-117, 1926.
Klein, A., Joplin, R. J., and Reidy, J. A.: The treatment of slipped capital femoral epiphysis, J.A.M.A. 136:445-449, 1948.
Klein, A., Joplin, R. J., Reidy, J. A., and Hanelin, J.: Slipped Capital Femoral Epiphysis, Springfield, Ill., Thomas, 1953.
Kocher, T.: Über Coxa Vara, eine Berufskrankheit der Wachstumsperiode, Deutsche Ztschr. Chir. 38:521-548, 1894; 40:411-426, 1895.
Löfgren, L.: Slipping of the upper femoral epiphysis, signs of endocrine disturbance, Acta chir. scandinav. 106:153-165, 1953.
Martin, P. H.: Slipped epiphysis in the adolescent hip, J. Bone & Joint Surg. 30A:9-19, 1948.
Maunder: Separation of the epiphyseal head of the femur, Lancet 1:192, 1870.
Maydl, K.: Coxa Vara und Arthritis Deformans Coxae, Wien. klin. Rundschau 11:153, 171, 187, 1897.
Mayland, A. E.: Traumatic separation of the epiphysis of the head of the femur, Brit. M. J. 1:709, 1892.
Monks, G. H.: A case of unusual deformity of both hip joints, Boston M. & Surg. J. 115: 468-470, 1886.
Moore, R. D.: Conservative management of adolescent slipped capital femoral epiphysis, Surg., Gynec. & Obst. 80:324-332, 1945.
Müller, E.: Über die Verbiegung des Schenkelhalses im Wachstumsälter, Beitr. klin. Chir. 4:137-148, 1888-1889.
Paré, Ambroïse: Fractures of the neck of the femur *in* Oeuvres complètes, vol. 2, book 13, chap. 21, Paris, 1840, and Cinq livres de chirurgie, Paris, 1572.
Perrin, M.: La coxa vara, Rev. d'orthop 23: 337, 441, 531, 1912.
Petit, J. L.: Maladies des os; description exacte des os, p. 354, Leyden, 1705. Also Traité des maladies des os, Oeuvres complètes, p. 169, Paris, 1837.
Poland, John: Traumatic separation of the epiphyses, London, Smith Elder & Co., 1898.
Pomeranz, M. M., and Sloane, M. F.: Slipping of the proximal femoral epiphysis; therapeutic results in 101 cases, Arch. Surg. 30:607-624, 1935.

Ponseti, I., and Barta, C. K.: Evaluation of treatment of slipping of the capital femoral epiphysis, Surg., Gynec. & Obst. 86:87-97, 1948.

Post: Separation of the epiphysis of the femur, New York M. J. 3:190, 1840.

Rammstedt, C.: Über die traumatische Lösung der Femurkopfepiphyse und ihre Folgeerscheinungen, Arch klin. Chir. 61:559, 1900.

Richerand: On fracture of the neck of the femur, Paris thesis, 1799.

Rognetta, M: Mémoire sur la divulsion traumatique des épiphyses, Gaz. méd. (de Paris), vol. 2, No. 28, pp. 433-438, 1834.

[illegible]

Rushmore, J. D.: Separation of femoral head, Tr. Am. S. A. 13:502, 1895.

Sabatier: Mémoires de l'Acad de chir., vol. 10, Paris, 1768.

Schlesinger, A.: Zur Aetiologie und pathologischen Anatomie der Coxa Vara, Arch klin. Chir 75:629, 1905.

Schultz, Julius: Zur Kasuistic der Verbiegungen des Schenkelhalses, Ztschr. orth. Chir. 1:49, 1892.

Smith, Noble: Separation of the epiphysis of the caput femoris, Lancet 1:538-539, 1886.

South, J. F.: A separation of the head of the thigh bone from the neck consequent on external violence, when ossification is incomplete, which is possible only in young subjects, Chelius' System of Surgery 1:565, 1847.

Spillman, E.: *In* Cuisse (pathologie) Dict. Encycl. des Sci. Med. 24:238, Paris, Masson, 1883

Sprengel, Otto G. K: Über die traumatische Lösung der Kopfepiphyse des Femur und ihr Verhältnisse zur Coxa Vara, Arch klin. Chir. 57:805-840, 1898.

Stetter. Centralbl. Chir. No. 36, 561, September 8, 1877.

Stovall, S. L.: Separation of the capital femoral epiphysis in the adolescent, Surg., Gynec. & Obst Internat. Abstracts 88:193-203, 1949.

Sturrock, C. A.: The after results of simple separation of epiphyses, Edinburgh Hosp. Rep. 2:598-604, 1894.

Sudeck, P.: Zur Anatomie und Aetiologie der Coxa Vara Adolescentium, Arch. klin Chir. 59:504, 1899.

Sutro, C J.: Slipping of the capital epiphysis of the femur in adolescence, Arch. Surg. 31:345, 360, 1935.

Wagner, L. C.: Wedge osteotomy of the neck of the femur in advanced cases of displaced [illegible] 1940.

Whitman, Royal: Observations on bending of the neck of the femur, in adolescence, with particular reference to the diagnosis and significance of the affection, New York M. J. 59: 769-773, 1894

———: Further observations on injuries to the neck of the femur in early life, with reference to the distinction between fracture of the neck of the femur and epiphyseal disjunction as influencing positive treatment, M Rec. 75: 1-7, 1909.

Wiberg, Gunnar: Epiphyseolysis capitis femoris, Acta orthop. scandinav. 12:179-213, 1941.

Wilson, P. D.: Displacement of the upper epiphysis of femur treated by open reduction, J.A.M.A. 83:1749-1756, 1924.

———: Treatment of slipping of the upper femoral epiphysis with minimal displacement, J. Bone & Joint Surg. 20:379-399, 1938.

Wright, G. A.: Hip Disease in Childhood, p 239, London, Longmans, 1887.

Wright, W., and King, Donald: The treatment of slipping femoral epiphysis (epiphyseolysis), Am. J. Surg. 91:894-899, 1956.

Zesas, Demis G.: *Die Coxa Vara und ihre* Beziehungen zu inneren Krankheiten, Zentralbl. Grenzgeb. Med. u. Chir. 7:833-845; 849-862; 896-918; 940-946, 1904.

Olisthese del Epiphyse Supero-Femoral

Summario in Interlingua

Le characteristicas clinic e le constatationes roentgenographic, le pathologia macro- e microscopic del tessutos molle como etiam de osso e cartilagine, e le curso natural de olisthese del epiphyse supero-femoral es ben cognoscite depost vinti-cinque annos. Le causa del disturbation vasculare del mollification al placa epiphysal que precede le

olisthese es non ancora clarmente definite. On ha demonstrate repetitemente in numeros significative de coxas que le separation del epiphyse ab le capite per medios chirurgic—manipulation, forte traction, o operation aperte—es sequite per necrosis aseptic del epiphyse, con dolores, limitation del motion, e invaliditate, in un si alte procentage del casos que tal manovras es justificate solmente sub le plus inusual conditiones. Il ha etiam essite monstrate que le resultatos del curation natural del morbo es frequentemente melior que le resultatos post reduction del olisthese e que necrose aseptic non occurre sin intervention chirurgic. In plus, immobilisation del epiphyse olisthetic—specialmente in extension, rotation interne, e abduction—causa un limitation permanente del mobilitate.

Epiphysiolisthese es a vices prevenibile per proteger le coxa sin immobilisation, plus certemente per fixation interne per medio de clavos o vites, o per le insertion de cavilias de osso a transverso le placa epiphysal. Fixation interne porta con se un certe (ben que basse) risco de necrosis aseptic con invaliditate consequente, durante que iste complication ha nunquam occurrite in un numerose serie de coxas tractate per caviliation de osso. Le melior resultatos es obtenite quando le morbo es recognoscite in su prime stadios de maniera que le olisthese pote esser prevenite.

Le prompte recognition del epiphysiolisthese depende del education del medicos qui primo vide le juvene patiente post le comenciamento del morbo. Portation de peso deberea esser eliminate si tosto que le presentia del morbo es suspicite. Le discoperta del exacte causa del morbo e de un methodo pro prevenir lo o al minus pro curar lo promptemente sin intervention chirurgic va esser le major contribution del futuro.

Ponseti, I., and Barta, C. K.: Evaluation of treatment of slipping of the capital femoral epiphysis, Surg., Gynec. & Obst. 86:87-97, 1948.
Post: Separation of the epiphysis of the femur, New York M. J. 3:190, 1840.
Rammstedt, C.: Über die traumatische Lösung *der Femurkopfepiphyse und ihre Folgeerscheinungen, Arch. klin. Chir. 61:559, 1900.*
Richerand: On fracture of the neck of the femur, Paris thesis, 1799.
Rognetta, M.: Mémoire sur la divulsion traumatique des épiphyses, Gaz. méd (de Paris), vol. 2, No. 28, pp. 433-438, 1834.
Rotter: Ein Fall von doppelseitiger rhachitischer Verbiegung des Schenkelhalses, Muench. klin. Woch. 37:32, 547, 1890.
Rushmore, J. D.: Separation of femoral head, Tr. Am. S. A. 13:502, 1895.
Sabatier: Mémoires de l'Acad. de chir., vol. 10, Paris, 1768.
Schlesinger, A.: *Zur Aetiologie und pathologischen Anatomie der Coxa Vara, Arch. klin. Chir. 75:629, 1905.*
Schultz, Julius: Zur Kasuistic der Verbiegungen des Schenkelhalses, Ztschr. orth. Chir. 1:49, 1892.
Smith, Noble: Separation of the epiphysis of the caput femoris, Lancet 1:538-539, 1886
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Spillman, E.: *In* Cuisse (pathologie) Dict Encycl. des Sci. Med. 24:238, Paris, Masson, 1883.
Sprengel, Otto G. K.: Über die traumatische Lösung der Kopfepiphyse des Femur und ihr Verhältnisse zur Coxa Vara, Arch klin. Chir. 57:805-840, 1898.
Stetter. Centralbl. Chir. No. 36, 561, September 8, 1877.
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Sturrock, C. A.: The after results of simple separation of epiphyses, Edinburgh Hosp Rep. 2:598-604, 1894.
Sudeck, P.: Zur Anatomie und Aetiologie der Coxa Vara Adolescentium, Arch. klin. Chir. 59:504, 1899.
Sutro, C. J.: Slipping of the capital epiphysis of the femur in adolescence, Arch. Surg. 31:345, 360, 1935.
Wagner, L. C.: Wedge osteotomy of the neck of the femur in advanced cases of displaced [illegible] 1940.
Whitman, Royal: Observations on bending of the neck of the femur, in adolescence, with particular reference to the diagnosis and significance of the affection, New York M J. 59: *769-773, 1894.*
———: Further observations on injuries to the neck of the femur in early life, with reference to the distinction between fracture of the neck of the femur and epiphyseal disjunction as influencing positive treatment, M. Rec. 75: 1-7, 1909.
Wiberg, Gunnar: Epiphyseolysis capitis femoris, Acta orthop. scandinav. 12:179-213, 1941.
Wilson, P. D.: Displacement of the upper epiphysis of femur treated by open reduction, J.A.M.A. 83:1749-1756, 1924.
———: Treatment of slipping *of the upper femoral epiphysis with minimal displacement,* J. Bone & Joint Surg. 20:379-399, 1938.
Wright, G. A.. Hip Disease in Childhood, p. 239, London, Longmans, 1887.
Wright, W., and King, Donald: The treatment of slipping femoral epiphysis (epiphyseolysis), Am J. Surg. 91:894-899, 1956.
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olisthese es non ancora clarmente definite. On ha demonstrate repetitemente in numeros significative de coxas que le separation del epiphyse ab le capite per medios chirurgic—manipulation, forte traction, o operation aperte—es sequite per necrosis aseptic del epiphyse, con dolores, limitation del motion, e invaliditate, in un si alte procentage del casos que tal manovras es justificate solmente sub le plus inusual conditiones. Il ha etiam essite monstrate que le resultatos del curation natural del morbo es frequentemente melior que le resultatos post reduction del olisthese e que necrose aseptic non occurre sin intervention chirurgic. In plus, immobilisation del epiphyse olisthetic—specialmente in extension, rotation interne, e abduction—causa un limitation permanente del mobilitate.

Epiphysiolisthese es a vices prevenibile per proteger le coxa sin immobilisation, plus certemente per fixation interne per medio de clavos o vites, o per le insertion de cavilias de osso a transverso le placa epiphysal. Fixation interne porta con se un certe (ben que basse) risco de necrosis aseptic con invaliditate consequente, durante que iste complication ha nunquam occurrite in un numerose serie de coxas tractate per caviliation de osso. Le melior resultatos es obtenite quando le morbo es recognoscite in su prime stadios de maniera que le olisthese pote esser prevenite.

Le prompte recognition del epiphysiolisthese depende del education del medicos qui primo vide le juvene patiente post le comenciamento del morbo. Portation de peso deberea esser eliminate si tosto que le presentia del morbo es suspicite. Le discoperta del exacte causa del morbo e de un methodo pro prevenir lo o al minus pro curar lo promptemente sin intervention chirurgic va esser le major contribution del futuro.

Section II

THE PATHOLOGIC PHYSIOLOGY OF METABOLIC BONE DISORDERS

A Symposium

(Conclusion of Section I, Clinical Orthopaedics No. 9)

EDWARD C. REIFENSTEIN, JR., M.D.
Guest Editor



16

The Long-Range Effects of Radiation on Bone

L. HENRY GARLAND, M.B.*

In 1903 Perthes demonstrated retardation of bone growth in experimentally irradiated chickens.[12] Some 6 years later Cluzet showed that callus formation following fracture could be delayed by irradiation.[5] Since that time there has been a mounting collection of experimental and clinical evidence to show the deleterious effects of heavy doses of irradiation on bone growth.[2,6,16]

In 1929 Martland[10] summarized the carcinogenic effects of large amounts of ingested irradiation in a careful report of osteogenic sarcomas that developed in watch-dial painters who had been using radioactive materials. Subsequent to that time a series of studies based on ingestion or injection of various artificial radioactive isotopes have appeared.

While these examples of arrested bone growth and late osteogenic sarcoma are of considerable general interest, a more common problem encountered by practicing physicians is that of bone necrosis following curative or radical radiotherapy to lesions such as carcinomas of the tonsil, the breast and female adnexa.[8,11,14] Intra-oral, mammary and uterine carcinomas are relatively common; vigorous external radiation of the primary tumor frequently is curative and is followed in a small percentage of cases by late radiation "osteitis," necrosis or fracture. Therefore, it is evident that there is a wide spectrum of late radiation effect in bone for consideration.

PATHOLOGY

The pathologic changes occurring in bone depend on several factors, notably the dose absorbed, the time required for delivering that dose, the volume of tissue irradiated, the metabolic status of the bone at the time of irradiation and the presence or the absence of associated lesions such as infection, neoplasm or trauma.[1,3,4,7] The calculation of absorbed dose in bone and cartilage presents many problems. Most radiation-dose measurements may be recorded in terms of rads or absorbed roentgens, delivered in a certain period of time to a certain volume. However, because of the increased scattering in calcium-containing tissue when voltages below about 500 KV are employed, the physical dose then absorbed in bone is believed to be almost double that in adjacent soft tissues. Conversely, in the megavoltage range, when using beams of 2 million volts and more, the dosage in bone is reported to be essentially similar to that in adjacent soft tissue As a result, it is not easy in reading reports of pathologic changes in the literature to estimate reliably the bone dose which produced those changes.

In clinical practice the long-range effects on bone are those of injury to the periosteum (with variable degrees of edema, fibrosis and avascularity), destruction of osteocytes, inhibition of osteoblast formation, obliteration of vascular channels and variable degrees of hyalinization and fibrosis. Growing cartilaginous or bony structures may show inhibition or arrest, depending upon the dose delivered. Heavily irradiated bone may undergo necrosis and sequestration.[9,13] Prior bony in-

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volvement by infection, neoplasm or injury will complicate or enhance the changes very greatly. Spontaneous development of metaplasia and malignant change may also result.

ROENTGENOLOGIC CHANGES

The late effects of radiation parallel the gross pathologic changes:

1. **Epiphysis.** The changes range from delayed growth to complete arrest of epiphyseal development.

2. **Diaphysis.** Roentgen examination may reveal areas of decreased density (so-called radiation osteitis), increased density (radiation sclerosis or necrosis) or neoplastic alteration. Radiation osteogenic sarcoma is grossly similar to spontaneously occurring sarcoma. In this connection it should be observed that *post hoc* is not always *proctor hoc*. For example, it is known that a small number of osteoclastomas or benign giant-cell tumors of bone eventually manifest malignant characteristics; the percentage given most frequently is 5. If a primary benign giant-cell tumor of bone is treated by irradiation with successful healing and recalcification, and shows sarcomatous changes 25 years later, who is to be certain that these are not spontaneous changes in some residual giant-cell-tumor elements? This comment applies to properly treated giant-cell lesions, and not to grossly over-irradiated ones, wherein there is reasonable suspicion that the radiation may be sarcomagenic.

DOSE AND LATENT PERIOD

Diagnostic Procedures. There are few reliable reports of serious late radiation effects on bone from properly performed roentgen diagnostic procedures. However, there are a few reports of osteitis from the early days of radiology, when very long exposures were required to secure roentgenograms of thick parts on insensitive glass plates.

Radiotherapy for Benign Lesions. There have been an extensive series of reports of epiphyseal arrest following large doses of radiation for hemangiomas occurring near growing cartilage. A small number of tragic cases of radiation osteitis in the calvarium following excessive and erroneous radiotherapy for tinea capitis have been reported. Finally, pituitary adenomas may be classified as intrinsically benign lesions: correct roentgen therapy for these frequently is successful in restoring vision and controlling endocrine changes. Many years after such roentgen therapy it is not unusual to have areas of decalcification in the temporal bones and the frontal bone through which the beams of therapy had been directed. This type of silent decalcification of localized areas of skull is asymptomatic and does not alarm the informed clinician.

Radiotherapy for Malignant Lesions. As indicated in the introduction, late radionecrosis of mandible, rib, pelvic girdle and some other structures must be regarded as an unavoidable occasional complication of properly given radiotherapy. By careful protraction of treatment and reasonable restriction of dosage, the incidence can be kept at a low figure. Furthermore, frequently the bone changes that develop are silent and detected only in roentgenograms of the area made at the time of periodic examination. Frequently the silent fractures of the pubic rami heal during a period of 6 to 12 months. Silent fractures of the femoral necks can be aided to heal by proper immobilization.

So far as epiphyseal arrest is concerned, we have seen no well-documented examples of small doses (for example, less than 300 r in one day to areas not over 15 cm. in diameter) having produced any late effects. Perhaps the most accurate attempt relating dose to growth disturbance is the study by Wittenborg and Neuhauser on irradiation effects of the spine in children.[15] They point out that in the treatment of neuroblastoma and Wilm's tumor there is unavoidable radiation to the growing spine; if less than 1,000 r is delivered to the spine, no late roentgen or clinical effects on bone have been demonstrated. However, almost in-

variably a tissue dose of over 2,000 r produces discernible bony alteration. This effect is related directly to the dose delivered to the spine and related inversely to the age of the patient.

It is difficult to predict the interval between irradiation and the development of osteitis or necrosis. It may be as short as 6 months or as long as 25 years. In our experience with some 200 patients treated for various types of intra-oral carcinoma, including tongue, tonsil, tonsillar pillar and oropharynx, we have had only two disabling cases of radionecrosis of the mandible, despite a reasonably satisfactory cure rate; the doses were in the neighborhood of 6,000 r into the primary tumor in a period of 4 to 6 weeks.

In an experience with some 600 patients with carcinoma of the breast, treated by preoperative, postoperative or combined methods of radiotherapy, we have had an incidence of about 10 per cent of silent radiation osteitis of the upper anterior ribs. The actual incidence may well be higher, but some of these women do not have roentgenograms of their chest made several years following treatment.

We have had an experience with some 350 women with carcinoma of the cervix, treated by full courses of external roentgen and internal radium therapy. In about 5 per cent of these women, variable degrees of radiation osteitis have been manifest in pubic rami or femoral necks. Only three have had clinical fractures requiring treatment.

Most of these bone changes were manifest between 3 and 10 years following treatment. However, in the case of the mandible, which has been the site of infection, and especially in the mandible in which teeth have not been removed prior to radiation therapy, earlier evidence of injury often is manifested.

We saw one patient in whom osteogenic sarcoma of the scapula developed 25 years after intensive radium therapy at another city. This therapy had been given for a giant-cell tumor of the acromion process, using long radium needles with light filtration. The patient had early pulmonary metastases and died within one year. Such heavy local radium therapy is no longer used for this lesion.

The problem of radiation changes following injection or administration of radioactive materials, either natural or artificial, is one occupying the interests of many scientists. Every worker in this field studies the classic report of Martland[10] and the later important reports of Aub, Evans *et al.*[1] Their cases were related mostly to the naturally occurring radioactive isotopes of radium. However, there is every reason to believe that comparable changes would occur following large amounts of any radioactive isotope that is deposited selectively in bone. As previously stated, the calculation of bone dose from injected radioisotopes is quite difficult. It has been estimated that some tumors have developed following doses as low as 2,000 r, but most apparently require larger doses. The latent period ranges from 5 to 25 years. The site of development of the lesions is varied, but there is a predilection for the mandible, the pelvis and the long bones of the leg.

The question of whether milk drinkers are ingesting too much radiostrontium (Sr^{90}) is currently under debate. Milk has always contained minute traces of radiopotassium; owing to nuclear bomb tests, it now contains radiostrontium as well. The long-range effects of significant doses of this radioisotope include anemia, leukemia and bone sarcoma. It is doubted that the present situation is hazardous.

DISCUSSION

The late effects of properly employed diagnostic roentgen radiation on human bone are negligible. The radiologist knows the output of his diagnostic apparatus, both radiographic and fluoroscopic; he knows the approximate dose in roentgens which bones receive whenever diagnostic exposures are made; he uses added aluminum filters of at

least 2-mm. thickness, adequate cones and *the minimum number* of exposures necessary to secure the information required to reach a diagnosis. He is taught that in the past many thoughtless persons, including professional men, kept their fingers in the mouths of patients while making diagnostic roentgenograms of the teeth and suffered irreparable damage to skin, soft tissues and bones. There is no excuse for such tragedies.

In radiotherapy of benign lesions, the radiologist uses his beams with discretion and directs the beam away from neighboring cartilage or bone if amounts approach those that may produce late changes. Usually, when such cartilage or growing bone cannot be shielded, he does not use radiotherapy. *In the treatment of malignant disease,* when cure is the aim, the radiotherapist *accepts a small percentage of late radiation* effects as unavoidable. The surgical cure of *many carcinomas carries* a small morbidity and mortality; similarly, the radiologic cure *of these lesions carries a small morbidity.* Careful placement of radiation, accurate aiming of beams and judicious spacing of treatments all help to reduce the incidence of late changes. *The fact that such can* develop after a latent period of many years must be stressed *continuously, since, unfortunately,* radium and other radiating sources are not restricted to those with adequate training and experience in their use. Radium can be rented by *almost any person in the* United States. Powerful x-ray apparatus is easily obtained. When either *radiant* source is employed in excessive or unduly protracted *amounts,* late effects may be anticipated. It is possible to cure intra-oral carcinoma *with doses of 6,000 r given in* a single course during a period of about 4 to 6 weeks. *The adjacent mandible or other* bony parts may receive doses in this range or lower. Serious late effects are uncommon. On the other hand, if 200 or 300 r is given intermittently, say twice a week, for 20 or 30 weeks, the total dose is still only about 6,000 r, but the chance of serious late radiation injury to bone is greatly increased. Apparently such intermittent radiation has a more harmful effect on *the endothelium of* small blood vessels and is more apt to be followed by *late fibrosis of deeper structures,* including marrow.

In summary, late radiation effects on bone range from minimal growth disturbance to *radiation-induced* sarcomas. In *the hands* of the inexperienced and untrained, the potential frequency of these undesirable effects is increased many times. The occasional *occurrence of radiation osteitis* as a result of radical radiotherapy for the cure of malignant disease is an unavoidable risk which should be understood and accepted in the same light as comparable risks following proper surgical management of patients with this disease. Long-range effects of radiation in bone can be minimized by careful utilization of all forms of diagnostic and therapeutic irradiation.

REFERENCES

1. Aub, J C, Evans, R. D, Hempelmann, L H., and Martland, H. S. Late effects of internally deposited radioactive material in man, Medicine 31:221, 1952.
2. Bisgard, J. D., and Hunt, H B : Influence [illegible] 19[illegible].
3. Cahan, W. G., *et al* · Sarcoma arising in irradiated bone, Cancer 1:3, 1948.
4. Camp, J D., and Moreton, R. D.: Radia*tion necrosis of calvarium, Radiology* 45: 213, 1945
5. *Cluzet. Quoted by Copeland,* Ref 6
6. Copeland, M. M. The effect of ionizing radiations on bone, Am Acad Orthop. Surgeons Instructional Course Lectures 10: 191, 1953
7. Ewing, J : Radiation osteitis, Acta radiol. 6:399, 1926
8. Jacox, H W.: Radiation effects on bone, Radiology 59:744, 1952
9. Lawrence, E. A : Osteoradionecrosis of mandible, Am J. Roentgenol *55:733,* 1946.
10. *Martland, H. S.:* Osteogenic sarcoma in dial painters using luminous paint, Arch. Path. 7:406, 1929.
11. Paul, L. W., and Pohle, E. A.: Radiation

osteitis of the ribs, Radiology 38:543, 1942.
12. Perthes: Quoted by Copeland, Ref. 6.
13. Rigler, L. G., Gratzek, F. R., and Holmstrom, E. G.: Post-irradiation bone changes, Am. J. Roentgenol. 53:62, 1945.
14. Stewart, M. B.: Osteoradionecrosis and cancer of the head and neck, Arch. Otolaryng. 38:403, 1943.
15. Wittenborg, M. H., Neuhauser, E. B. D., *et al.*: Irradiation effects of roentgen therapy on the growing spine, Radiology 59: 637, 1952.
16. Woodard, H. Q, and Coley, B. L.: Correlation of tissue dose and clinical response in irradiation of bone tumors and of normal bone, Am. J. Roentgenol. 57:464, 1947.

Effectos a Longe Vista Exercite Super le Ossos per Lor Irradiation

Summario in Interlingua

Le effectos a longe vista que le radiation exerce super le ossos depende del dose de rads absorbite, del tempore requirite pro le application de ille dose, del volumine de tessuto irradiate, e del stato metabolic del osso al tempore del irradiation. Altere factores que exerce un influentia super le effectos tardive include infection, trauma, e neoplasma. Le calculation del dose absorbite in osso e cartilagine non es simple. Illo deveni specialmente complexe in le caso de administrationes interne de isotopos radioactive.

In le practica clinic, le pathologic effectos a longe vista es insultos del periosteo, destruction de osteophytos, inhibition del formation de osteoblastos, oblitteration de canales vascular, e varie grados de hyalinisation e de fibrosis. Post dosages adequate, structuras cartilaginose o ossee exhibi varie grados de inhibition o de arresto. Post multo forte doses, ambe le mentionate sitos exhibi necrosis o sequestration. Metaplasia e neoplasia maligne pote evenir in le curso del tempore.

Le alterationes roentgenologic consiste de varie grados de discalcification, de sclerosis, de atrophia, e—in le presentia de certe typos de neoplasia—de neoformation ossose.

Le effectos tardive del correcte empleo diagnostic de roentgeno-irradiation de ossos human deberea esser negligibile. Tamen, post irradiation therapeutic, specialmente in le tractamento de certe typos de neoplasma, un basse procentage de tardive effectos radiational es inevitabile e debe esser acceptate como parte del morbiditate de radiotherapia correcte. Le intervallation judiciose del doses, le evitar de forte grados de radiation in le presentia de infection, e le evitar de trauma durante le periodo de osteoporosis—omne iste mesuras pote contribuer a un reduction del serietate del effectos tardive. On sperava que megavoltages e le uso de fasces ab cobalt radioactive resultarea in un reduction significative del incidentia de non-expectate osteonecrose, sed isto ha non occurrite.

Le diagnose differential de osteitis radiational, infection, e primari o metastatic morbo del osso pote esser extrememente difficile. Illo require le consideration meticulose de omne aspectos del caso ante le completion del diagnose radiologic.

17

The Use of Corticosteroids in the Treatment of Painful and Stiff Shoulders

T. B. QUIGLEY, M.D.*

Six years ago when the supply of corticotropin became great enough to include in clinical study what might be called nuisance conditions as well as the more serious collagen and degenerative diseases, an investigation of its effect on painful shoulders was begun at the Peter Bent Brigham Hospital. All patients with painful shoulders, whatever the cause, were seen, excluding only those whose shoulder symptoms were part of a generalized disease. All the usual causes for shoulder pain were encountered, including inner cuff tendon tears of varying degree, extrinsic neuromuscular lesions of various types, acromioclavicular abnormalities, a few tumors and cysts, and a few previously unrecognized minor fractures. For these, standard methods of treatment were carried out with standard results.

But most of the patients—and by far the most interesting—fell into two groups: those whose pain and limitation of motion were caused by a sterile inflammatory reaction to *a deposit of calcium at or near the greater* tuberosity of the humerus; and those whose limitation of motion and pain were caused by a contracture of soft tissues at the shoulder joint.

In the first group it was reasoned that since there was little doubt that an inflammatory process was causing the patient's symptoms (albeit a somewhat mysterious inflammatory process, since no bacteria had ever been·incriminated), the antiphlogistic properties of the hormones secreted by the adrenal gland when stimulated by corticotropin might be beneficial. Accordingly, 6 patients were admitted to the hospital, surveyed carefully for contraindications and given corticotropin. The following history of the first patient so treated on April 9, 1951, is typical:

Case 1. A 45-year-old housewife complained of dull, gnawing pain at the right shoulder of 4-weeks' duration. There was no history of injury. Active and passive abduction was limited to 10°, external rotation to 35° and internal rotation to 90°. There was moderate tenderness over the greater tuberosity of the humerus, and roentgenograms disclosed a small calcific deposit in the inner cuff tendons at this point. Procaine was injected into the soft tissues about the greater tuberosity with immediate relief, which, however, was only temporary. A week later the patient returned to the hospital, having fainted from the severity of the pain. She was admitted, surveyed for contraindications to corticotropin, none was found, and she was given 10 mg intravenously over an 8-hour period in saline. Pain disappeared within a few hours, before the in- [illegible] but again were suppressed by 30 mg of corticotropin administered intravenously. There has been no recurrence in 5 years.

All 6 patients were relieved similarly, more or less dramatically, depending on the

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FIG. 1. Case 1. *(Left)* Severe acute calcific tendinitis. Pain restricts abduction and is reflected in the patient's posture. *(Right)* Four hours after receiving half of an intravenous infusion containing 10 mg. of corticotropin.

severity of symptoms.[17] It was observed that the shorter the duration of symptoms, the more severe the pain, and the greater the tenderness, the more effective was corticotropin in suppressing the inflammatory response and restoring the shoulder to a normal, painless range of active motion.

When hydrocortisone acetate became available in a form suitable for direct parenteral use, its advantages were immediately apparent. Hospitalization was no longer necessary. Contraindications to corticotropin could be forgotten, and the great majority of patients with acute calcific tendinitis could be relieved promptly by a simple office or outpatient procedure. Our original impressions were confirmed promptly, and for the past 4 years the treatment of choice for acute calcific tendinitis of the inner cuff tendons of the shoulder has been, basically, the injection of hydrocortisone acetate into the calcific deposit. This apparently has been a general experience, but it has not been documented extensively in the literature.[3,6,14,18-20]

Therefore, it seemed worth while to review 25 consecutive personal cases of acute calcific tendinitis treated between June, 1955, and October, 1956, and followed to March, 1957.

The group consisted of 9 males presenting 8 involved right shoulders and 1 left, and 16 females with 10 right and 6 left shoulders affected. The average age was 48.4 years, the oldest patient being 74 and the youngest 33. Eleven had symptoms for 1 week or less, 6 between 1 week and 1 month, 4 between 1 and 6 months, and 4 between 6 months and 2 years. Subjective pain and objective tenderness were practically equal in each case and were graded on a scale of 1 to 4 in severity. Five were classified as 1, 9 as 2, 7 as 3, and 4 as 4.

The more acute the symptoms and signs, the shorter their duration. All the patients in the Grade 4 category had suffered for less than 3 days. Nineteen of the 25 patients were relieved immediately by injection and have remained symptom free to the present time. Three were improved at a second visit 7 to 10 days after injection and refused a second injection. One was not relieved by the first injection and was relieved completely by a second. Another was partially relieved by the first injection and completely relieved by the second. Only 1 patient's pain and tenderness failed to respond to 2 injections.

There were no complications; in fact, no complication has occurred in at least several hundred injections of hydrocortisone at the Peter Bent Brigham Hospital in the past 5 years.

The procedure used in this series for the past 3 years has been the injection of a mix-

ture of 25 to 50 mg. of hydrocortisone acetate, 150 units of hyaluronidase and 6 to 10 cc. of 1 per cent procaine. The inner cuff tendons are distended thoroughly with this fluid through several 18 or 20 gauge needle punctures focused at the point of greatest tenderness, which is almost invariably the point at which the calcium is seen to lie in the roentgenogram. This seems to accomplish three things: (1) the capsule enveloping the calcific mass is decompressed mechanically by the needle punctures; (2) the subdeltoid bursa is somewhat stretched, and the tissues of the inner cuff tendons themselves are distended by the injected fluid; (3) the hydrocortisone is disseminated throughout the inflamed tissues to exert its anti-inflammatory effect to greatest advantage. Almost invariably, return of symptoms to a minor degree occurs a few hours after injection as the effect of the procaine subsides. Within 2 to 5 days at the latest, relief or improvement follows. Hyaluronidase was added to the mixture at first on purely empiric grounds but has since been supported by the work of Glassman *et al.*[4] These studies show that the dissemination of trypan blue (the molecule of which is larger than that of hydrocortisone) in the presence of a sterile inflammatory reaction is enhanced considerably by hyaluronidase. Certainly, all observers have agreed that the clinical results have been enhanced by the addition of hyaluronidase to the mixture.

Before hydrocortisone was available, roentgen therapy was felt to be the most effective and efficient treatment available. But the clinical superiority of hydrocortisone injections is now clear, and the disadvantage of adding even small increments to the patient's total lifetime roentgen dosage is avoided. We have had no experience with diathermy or ultrasonic methods of producing deep heat The studies of Murphy *et al*[12] have shown that none of these modalities with wavelengths between infrared and 1,600 cm raises the temperature of the tissues, even at depths of 3.0 cm., more than 2°. We have been impressed by the clinical observation that simple hot wet packs are as effective as any other source of heat in the management of stiff and painful shoulders, and no source of heat can be expected to do more than produce a nonspecific hyperemia and subjective sense of comfort. Very few patients with painful shoulders treated by hydrocortisone injection have required any sort of heat.

The injection of hydrocortisone or, for that matter, anything into the human body is not without its dangers as the following 2 cases will attest:

Case 2. A 70-year-old widow, who was in good health except for moderately severe, rather poorly controlled diabetes, visited her family physician because of pain in the right shoulder on movement. There was no history of injury. A diagnosis of "bursitis" was made, and 150 mg. of cortisone per day was given orally for the next 9 days. This did not produce relief within 2 or 3 days, and procaine was injected into the shoulder on several occasions. After these injections her shoulder became rapidly swollen, tender and more painful. Nine days after the onset of symptoms the patient was admitted to the Peter Bent Brigham Hospital in coma, with a temperature of 103, which rose within 48 hours to 105.5.

Shortly after admission a subdeltoid abscess was drained under local anesthesia of 600 cu. cm. of pus Despite heroic efforts to control her diabetes and very large doses of antibiotics to which the *Streptococcus viridans* and the *Streptococcus zymogenes* cultured from the wound were sensitive, death from septicemia occurred on the 7th day after admission to the hospital and the 16th day after the patient first complained of pain in the left shoulder.

Case 3. A middle-aged housewife in good general health experienced pain and tenderness on the anterior aspect of her shoulder while traveling abroad A cortisone compound and an oily preparation of prednisolone were injected into the tender area. Thirty-six hours later, in another European city, the shoulder pain was very acute, and 48 hours after the injection the shoulder joint was explored. No gross infection was encountered, but a culture was taken which subsequently produced a penicillin-resistant staphylococcus. Shock and clinical evidence of septicemia followed this operation. Erythromycin was given, and the septicemia was con-



Fig. 2. A very large, dense calcific deposit in the inner cuff tendons in a small woman which required removal to permit normal shoulder motion.



Fig. 3. True calcific bursitis. The inflamed calcific deposit has burst into the subdeltoid bursa which is outlined as a milky opacity.

trolled. The shoulder joint still was draining 6 months later.

The two tragedies can be considered only as iatrogenic. In each case the infection was introduced by the needle. It must not be forgotten that from the bacteriologic point of view, arthrocentesis is the same as arthrotomy, and the same basic precautions must be observed. There is no substitute for careful skin disinfection and scrupulous mechanical cleansing of needles and syringes. The shortcomings of boiling as compared with autoclaving are well known. Walter[21] has pointed out the real possibility of bacterial contamination which is inherent in the multiple-dose vial of biologicals and other preparations. It is to be hoped that manufacturers will supply injectable substances in single-dose packages.

Surgical removal of calcific deposits is still occasionally necessary, when the size of the mass interferes mechanically with shoulder motion or its hardness resists dissemination with the needle. Such a case is the following:

Case 4. A small, active mother of 4 children experienced several episodes of annoying to severe right shoulder pain over a 4-year period. Injection of hydrocortisone on two occasions produced only moderate relief. Some degree of pain and tenderness was always present, particularly on abduction beyond 60°. At this point a definite "catch" in motion was present, and grating could be felt beneath the acromion.

Roentgenograms disclosed a very large deposit of calcific material at the greater tuberosity, which by its size alone could easily explain the "catch" and grating. (Fig. 2.)

The distal third of the acromion was removed, the coraco-acromial ligament was divided, and several very large, dense calcified plaques were removed and their sites obliterated with fine interrupted catgut sutures.

Convalescence was uneventful, a guided exercise program was followed faithfully, and normal painless motion was present 3 months later. No further symptoms have occurred in 3 years.

A few patients describe increasing pain for several days followed by a sudden remission. Tenderness is diffuse over the deltoid, and, if the muscle is thin, fluctuation beneath it may be discernible. Roentgenograms (Fig. 3) show the entire subdeltoid bursa outlined by milky opacity. Presumably the inflamed calcific deposit in these cases has burst into the bursa and has produced a true bursitis with effusion. Aspiration and irrigation through 2 large-bore needles, followed by instillation of hydrocortisone acetate, hyaluronidase and procaine,

are very effective and are, in fact, the only situation for which this 2-needle technic is indicated in our experience.

The second large group of patients whose symptoms were associated with contractures of soft tissues about the shoulder joint were at first classified as "frozen" shoulder. However, it soon became apparent that this was too inclusive a term, of no greater precision than "back strain," "surgical belly" or "Pott's fracture," including a rather wide spectrum of causative factors, duration of symptoms, pain and limitation of motion. As experience increased, it became clear that a considerable number of these "frozen" shoulders fell into a standard pattern of symptoms and signs. These patients responded well to manipulative rupture of contracture of the antero-inferior aspect of the shoulder joint and suppression of post-manipulation pain by corticosteroids, and the syndrome has been labeled checkrein shoulder.[15,16]

The syndrome consists of a history in a middle-aged person of several weeks of disuse of a shoulder imposed by an episode of inflammation ("bursitis"), a minor injury, sometimes a myocardial infarct, but often no initiating episode at all. Gradually, the most commonly used lower ranges of shoulder motion return, and the less frequently used upper ranges of motion are checked. On physical examination both active and passive motion are sharply limited at about 45° of abduction and 50 per cent of the normal range of internal and external rotation. Roentgenograms usually are not remarkable, except occasionally, showing demineralization from disuse and in 16 per cent of cases calcific deposits at or near the greater tuberosity which are not in themselves painful or tender.

Etiology is not clear. Generalized disease is not associated with the syndrome, major trauma is rarely a factor, and rupture of the supraspinatus tendon and other elements of the rotator cuff has been established as unassociated with stiff shoulders.[9] The pathology of "frozen" shoulder can be as various as the degree of limitation of motion and pain which the patient presents. Adhesions across the subdeltoid bursa, generalized "capsulitis" and adhesion of the tendon along the biceps can all be present. However, the anatomic abnormality of checkrein shoulder has been well described by Neviaser[13] as adherence of the antero-inferior folds of the joint capsule to each other and to the adjacent glenoid and humerus. Neviaser's studies were based on observation of 15 stiff shoulders manipulated after surgical exposure of the joint. His findings have been confirmed in one instance in the present series in which the rigidity of the shoulder was so great that attempts at manipulation were abandoned, and open division of the contracture subsequently was carried out.

The generally accepted treatment of stiff "frozen" shoulders has been gradual stretching by active exercise and traction, together with appropriate heat, repeated procaine infiltration and, at times, roentgen radiation. This program is effective and safe, but it may require months and makes considerable demands on the patient's fortitude.

Manipulation with the patient under anesthesia has been almost universally condemned as dangerous and futile.[2,8,11] Pain after manipulation usually is so severe that active motion is difficult or impossible. By the time pain has subsided, adhesions and contractures can reform at times with more limitation of motion than before manipulation.

It seemed reasonable to assume that pain following the stretching or the tearing of soft tissues would be suppressed by corticosteroids. This has proved to be the case. In no case in the present series has medication other than codeine and aspirin been required to control pain after manipulation

As in the first group of patients with pain produced by acute inflammation of a calcific deposit, our first patients were given corticotropin either in a slow intravenous infusion or as a gel. This was soon abandoned in



FIG. 4. (*Left*) Incorrect method of manipulation of a "checkrein" shoulder. Fracture, dislocation and stretch injury to the neurovascular bundle are imminent. (Quigley, T. B.: New England J. Med. 250:188-192)

(*Right*) Correct method of manipulation of a "checkrein" shoulder. Force is applied with gentleness. The operator's forearm presses equally at all points along the patient's humerus, and his opposite hand depresses the scapula and the clavicle. The humerus is not used as a lever. (Quigley, T. B.: J.A.M.A. 161:850-854)

favor of oral cortisone, which was found to be equally effective. More recently, the injection of hydrocortisone acetate, hyaluronidase and procaine into the shoulder joint and hematoma formed by the ruptured structures has been carried out immediately after manipulation. This has proved to be quite satisfactory in suppressing pain, particularly when blood from the hematoma can be aspirated from the needle.

The manipulation itself is carried out with the patient under light thiopental (Pentothal)-nitrous oxide anesthesia and is a procedure not to be considered lightly, being fraught with obvious potential dangers. Gentleness in the application of force is essential. The patient's arm cannot be used as a lever. (Fig. 4.) The humerus is gently but firmly abducted on the scapula through the 10 to 20° occupied by the checkrein contracture. A tearing noise usually can be heard, and a sudden release of resistance is felt, followed by a free range of abduction. Further gentle manipulation in external rotation may be necessary, but no attempt is made to increase the range of internal rotation lest atrophic contracted rotator cuff muscles and tendons be torn. A needle then is introduced into the antero-inferior aspect of the joint, and aspiration is carried out until hematoma is encountered. A mixture of 100 mg. of hydrocortisone acetate, 450 units of hyaluronidase and 10 to 15 cc. of 1 per cent procaine next is injected into the hematoma and the joint.

An intensive program of active motion begins as soon as the patient recovers from the anesthetic, continues throughout the patient's stay in the hospital for the next 4 or 5 days, and concludes in instruction in a 15-minute pattern of exercise which the patient is advised to continue once daily indefinitely.

Sixty-six manipulations have been carried out on 64 shoulders in 61 patients who have received corticosteroids to suppress post-manipulation pain. In 3 patients both shoulders were manipulated, in 1 case on 2 occasions. The follow-up has varied from 5 years to 3 months. Of these 64 shoulders, 58 satisfied the criteria for the diagnosis of checkrein shoulder. Forty-one of these promptly regained and maintained normal active and passive motion. Seventeen were improved, retaining some degree of limitation of motion or pain, or both. The remaining 6 patients were all manipulated early in the

series, did not present a standard pattern of pain or limitation of motion and did not, therefore, fall into the group of checkrein shoulders. Their shoulders were either unimproved or made worse by manipulation. Complications consisted of one instance of sensory disturbance of the ulnar nerve, attributed to too vigorous manipulation in which recovery occurred after several months, one psychosis apparently precipitated by corticotropin which was successfully treated by psychotherapy, and one fracture of the anatomic neck of the humerus in a middle-aged housewife whose limitation of motion followed prolonged immobilization for fracture of the clavicle and was much more severe than the standard checkrein pattern.

One patient whose checkrein limitation followed a severe injury and whose limitation resisted attempts at manipulation was operated upon. A mass of myositis ossificans traumatica was removed from the subscapularis tendon and anterior capsule, and manipulation was carried out under direct vision, confirming Neviaser's findings.

It seems to be clear that those shoulders in which the contracture resists manipulation and those in which limitation of motion does not fit the checkrein pattern should have their contractures divided surgically after a fair trial of traction, guided exercise and other nonoperative measures.

The average duration of symptoms prior to manipulation was 7 months. The majority of patients had received previous treatment, including massage, heat, diathermy, ultrasound, procaine block of the cervical sympathetic chain and a considerable gamut of intramuscular injections, including vitamins, iron, corticotropin and other unspecified substances—all without relief. Many were described as "psychosomatic," but psychiatric symptoms disappeared promptly as normal painless motion of the shoulder returned. It would appear that the "periarthritic personality"[1] is at least as often the result of the painful stiff shoulder as its cause.

Another group of patients had been diagnosed as "shoulder-hand syndrome." In these also the stiff edematous hand and forearm returned promptly to normal as shoulder motion was restored. This experience has been confirmed by Moberg,[10] and it would appear that in many instances of "shoulder-hand syndrome" the simple progression of events of dependency plus disuse producing edema, and edema plus time-producing periarticular stiffness, explains the clinical picture.

Lyons[7] and Hayes[5] have called attention to the occurrence of postoperative adrenal exhaustion months or even years after administration of corticosteroids. This has not occurred in the present series of either acute calcific tendinitis or checkrein shoulder cases, probably because the total dosage both in amount and time was small. It seems a most unlikely possibility following one local injection of no more than 100 mg. of hydrocortisone acetate.

Note: Hyaluronidase (Wydase) used in this study was provided by the Wyeth Laboratories.

REFERENCES

1. Coventry, M. B.: Problem of painful shoulder, J.A.M.A. 151:177-185, 1953.
2. DePalma, A. F.: Surgery of the Shoulder, p. 147, Philadelphia, Lippincott, 1950.
3. Freeman, B. L., Jr.: Experiences with hydrocortisone, J. South Carolina M. A. 49:12, 1953.
4. Glassman, Jerome M., Blumenthal, Albert, Beckfield, William J., and Seifter, Joseph: Effect of hyaluronidase on inflammation, Proc. Soc. Exper. Biol. & Med. 82:323-328, 1953.
5. Hayes, M. A.: Surgical treatment as com- [illegible] steroid [illegible]
6. Hollander, J. L. [illegible] hydrocortisone in arthritis and allied conditions, J. Bone & Joint Surg. 35A:499, 1953.
7. Lyons, Champ: Personal communication.
8. McLaughlin, H. L.: On "frozen" shoulder, Bull. Hosp. Joint Dis. 12:383-393, 1951.

9. McLaughlin, H. L., and Asherman, E. G.: Legions of musculotendinous cuff of the shoulder; some observations based on results of surgical repair, J. Bone & Joint Surg. 33A:76-86, 1951.
10. Moberg, E.: The shoulder-hand-finger syndrome as a whole, Acta chir. scandinav. 109:284-292, 1955.
11. Moseley, H. F.: Shoulder Lesions, p. 142, Springfield, Ill., Thomas, 1945.
12. Murphy, Alma J., Paul, M. D., and Hines, H. M.: A comparative study of the temperature changes produced by various thermogenic agents, Arch. Phys. Med. 31: 151-156, 1950.
13. Neviaser, J. S.: Adhesive capsulitis of the shoulder: study of pathological findings in periarthritis of shoulder,, J. Bone & Joint Surg. 27:211-222, 1945.
14. Orbach, E. J.: Treatment of bursitis by local injection of hydrocortisone acetate, J. Internat. Coll. Surgeons 18:159-163, 1952.
15. Quigley, T. B.: Checkrein shoulder; a type of "frozen" shoulder; diagnosis and treatment by manipulation and ACTH or cortisone, New England J. Med. 250:188-192, 1954.
16. ———: Treatment of checkrein shoulder by manipulation and cortisone, J.A.M.A. 161:850-854, 1956.
17. Quigley, T. B., and Renold, Albert E.: Acute calcific tendinitis and "frozen" shoulder; their treatment with ACTH, New England J. Med. 246:1012-1014, 1952.
18. Ramsey, R. H., and Key, J. A.: Use of hydrocortisone (compound F) by local injection in arthritis and allied conditions, Missouri Med. 50:604, 1953.
19. Schneeberg, N. G.: Cortisone in the treatment of bursitis, J. Albert Einstein M. Center (Philadelphia) 1:142-145, 1953.
20. Steinbrocker, O.: Keeping the patient with a shoulder syndrome employable, Indust. Med. & Surg. 22:314-316, 1953.
21. Walter, Carl: Personal communication.

Le Uso de Corticosteroides in le Tractamento de Spatulas Dolorose e Rigide

Summario in Interlingua

Es presentate le resultatos de un studio sexenne del effectos de corticosteroides in le tractamento de spatulas dolorose e rigide. Le sequente conclusiones pare justificate:

1. Le injection local de un mixtura de acetato de hydrocortisona, hyaluronidase, e procaina es le melior therapia nunc disponibile pro acute tendinitis calcific. In un serie specimen de 25 casos consecutive, 24 monstrava un prompte alleviamento del dolores e del limitation de mobilitate e manifestava nulle complicationes. Tamen, le manovra non es totalmente innocue, viste un caso de morte per sepse in un patiente tractate alterubi Excision chirurgic remane le tractamento de election pro certe grande e dense depositos.

2. Inter le rigide e dolorose spatulas "congelate," un nove syndrome ha essite definite sub le nomine de *checkrein shoulder*, i.e. "spatula a brida false." Iste syndrome responde ben a manipulation e suppression delores post-manipulatori per administrationes de corticosteroides. Sexanta-sex manipulationes esseva effectuate in 64 spatulas. Cinquanta-octo de iste casos satisfaceva le criterios del diagnose de spatula a brida false. In 41, le stato normal esseva promptemente restaurate, e 17 se monstrava meliorate. In sex casos, que non correspondeva al criterios de spatula a brida false, nulle melioration esseva notate. Quasi omne le complicationes del serie total occurreva in iste gruppo. Tal casos deberea esser tractate per division chirurgic del structuras contrahite, post que mesuras non-operatori ha essite probate adequatemente.

18

Calcium Metabolism in Relation to Metastatic Malignancy*

William H. Baker, M.D.

Bone is a very frequent site of neoplastic metastasis and is surpassed in this respect only by the lungs and the liver.[59] In 1891, von Recklinghausen presented his now classic pathologic analysis of metastatic cancer in bone. By detailed study he indicated (1) that metastatic growths of bone appeared first within the bone marrow; (2) that they were blood borne; (3) that subperiosteal extensions appeared chiefly where main foramina traversed the cortical bone; and (4) that the order of frequency in different parts of the skeleton was as follows: vertebrae—proximal ends of femur, pelvis, ribs, sternum; proximal ends of the humerus, skull and other bones less frequently. All these conclusions have withstood the test of time and are as valid now as they were then.[53]

Although any carcinoma may metastasize to bone, the most frequent are those originating in breast, lungs, prostate, kidney and thyroid.[59] In carcinoma of the breast the incidence of skeletal metastasis in disseminated disease varies anywhere from 50 to 75 per cent.[24,59] In a recent study by bone-marrow analysis on consecutive cancer patients admitted to a cancer hospital, 10 per cent of all bone-marrow aspirations were positive for cancer cells, and in 37 per cent of these there was no radiologic evidence of tumor metastases.[33] The presence or the absence of tumor cells, as indicated by this technic, may prove ultimately to be of prognostic value.[35] Since a single bone-marrow aspirate is only a small sample, it is quite obvious that many more patients than these 10 per cent have carcinoma cells within their marrow cavity during life. It is also probable that subsequently these circulating tumor cells within the marrow cavity may be destroyed by an adverse host reaction. The prevalence of tumor cells within the marrow cavity must be due in large part to its rich vascular supply with trapping of blood-borne tumor cells. Extensive venous anastomoses from the inferior vena cava to the lumbar spine, as demonstrated by Batson,[9] must play a role in frequency of metastatic disease in that area, especially with primary tumors of the pelvic organs. Finally, the biochemical nature of the soil, which normally supports active marrow proliferation and cellular reproduction, constitutes a fertile area for proliferation and reproduction of tumor cells. As tumor cells encroach upon the red marrow, new areas of marrow production within the yellow or fatty marrow may develop, and thus ultimately the mid-portion of the long bone of the shafts becomes areas of increased vascular supply. The amount of new marrow produced usually is inadequate to compensate for the

* [illegible] ton, Mass This is publication No 907 of the Cancer Commission of Harvard University. This investigation was supported by a National Cancer Institute Research Grant, No. C 2421.

shortened life span of the red blood cells, and chronic anemia develops. That defective synthesis and formation is not a major factor is indicated by the increased erythropoiesis in remaining marrow as measured by Fe^{59}.[32,34]

As bone is destroyed it is replaced by connective-tissue stroma, and large numbers of multinucleated cells resembling osteoclasts can be seen at the site of the bone destruction.[59] The usual host reaction to such bone resorption is that of bone formation or repair, so that in most cases a mixture of osteolytic and osteoblastic reaction is seen. If bone destruction exceeds bone production, a net loss of calcium occurs, which is reflected by an increased excretion of calcium, primarily in the urine but also in the feces. In those tumors evoking a marked osteoblastic reaction, when bone production exceeds bone destruction, such as prostatic carcinoma and an occasional case of breast carcinoma, calcium excretion is decreased and may be lower than normal. Indeed, in some cases of prostatic carcinoma, with serum calcium of 9 to 10 mg. per cent, urinary calcium excretion after effective hormonal therapy may be as low as 5 to 10 mg. per 24 hours (normal range of 100-200 mg. per 24 hrs.).[6]

Metabolic data show that parallel increases and decreases of urinary phosphorus accompany the calcium changes, most of which can be accounted for by bone destruction or production.[39,40]

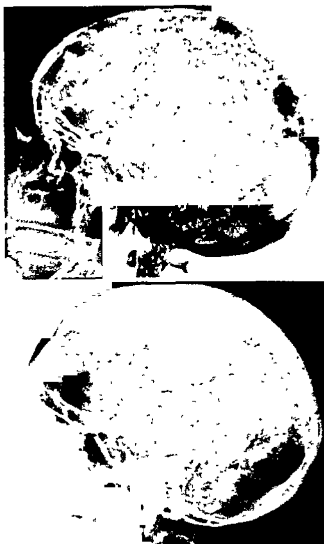


Fig. 1. M. S., 56. Osteolytic and osteoblastic involvement of skull due to primary breast carcinoma. *(Top)* Before Treatment. *(Bottom)* Ten months after testosterone propionate, 50 mg. intramuscularly 3 times per week. Note restoration of bone structure. (Nathanson, I. T.: Abstracts of Lectures, Fifth International Post Graduate Assembly in Endocrinology and Metabolism, p. 69)

OSTEOLYTIC VS. OSTEOBLASTIC METASTASES

In general, metastatic bone cancer produces a roentgenographic picture of osteolytic and osteoblastic reaction, but all variations exist (Fig. 1). Metastatic carcinoma of the prostate usually evokes a predominantly osteoblastic lesion (Fig. 2) whereas carcinoma of the thyroid characteristically produces an osteolytic type of lesion. In carcinoma of the prostate, the osteoblastic reaction may be so pronounced that hypocalciuria and hypocalcemia occur. Intravenous administration of radioactive calcium (Ca^{45}) to such patients is associated with an increased retention of administered radioactivity with increased exchangeable 9-day pool.[8]

In general, a slow-growing tumor will evoke a desmoplastic reaction in soft tissue and an osteoblastic reaction in bone. These two reactions are characteristic of the body's



FIG. 2. I. C., 65. *(Top)* Extensive osteoblastic metastases from primary prostate cancer. *(Bottom)* Marked improvement and restoration of bone structure 34 months after castration. (Nathanson, I. T., *Abstracts of Lectures, Fifth International Post Graduate Assembly in Endocrinology and Metabolism*, p. 111)

reaction to injury, which in the case of fracture is callus, and in soft tissue, scar formation. A recent pathologic analysis of metastatic cancer in bone indicated that the differences in bone response to various types of neoplasm were quantitative and not qualitative. The authors believed that the type of reaction produced was due mainly to rate of tumor growth and not to any abnormal substance produced by the cancer cell.[45] Since slow-growing tumors of the thyroid produce an osteolytic lesion, biochemical characteristics of the tumor cells must play an important part in determining the type of host reaction in bone. One of the many possible governing biochemical factors could be the high level of acid phosphatase which is found in prostatic carcinoma cells.[20] Acid phosphatase also has been demonstrated in carcinoma cells originating in the breast,[42,43] which produce a combination of osteolytic and osteoblastic metastasis and, in an occasional case, a complete osteoblastic reaction. Carcinoma cells of the thyroid and the kidney do not contain such concentrations of this enzyme, and, in addition, they are notoriously poor in stroma, thus failing to provide a matrix for calcium deposition It is obvious that much more biochemical knowledge is needed before we can determine the factors responsible for the differing bone response to different tumors. In most cases the host reaction to invading tumor cells is an attempt to confine and slow down the growth. Eventually this defense is overcome, and replacement of bony tissue by neoplastic cells results in a release of calcium and phosphorus in the blood stream, which is then excreted predominantly by the kidney.[6,17,39,40] If the released calcium is not utilized in a repair reaction of the host, it will exceed the capacity of the kidney to excrete such large quantities, and hypercalcemia and its attendant symptomatology ensue. In most cases the resultant hypercalcemia is associated with a normal or an elevated serum phosphorus; rarely with a low serum phosphorus, particularly when severe cachexia is present. When a low serum phosphorus accompanies the hypercalcemia, the blood-serum values are identical with those of hyperparathyroidism,[3] and there is difficulty in exact diagnosis.[3,35]

Albright *et al.*[3] observed such blood-serum values in a patient with renal cell carcinoma and a single metastasis to the ilium. Following roentgen therapy to the ilium, the blood-serum values returned to normal but with recurrence of the tumor

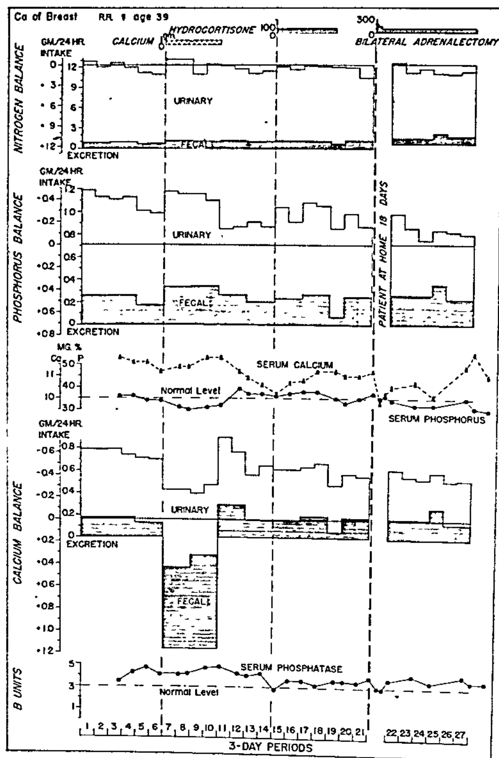


FIG. 3. Metabolic balance data in breast cancer. Marked negative calcium and phosphorus balance with hypercalcemia. Note that addition of 1.0 Gm. calcium levulinate orally caused less negative calcium balance. There was increase in fecal calcium with no change in urinary calcium, with net retention of 200 to 250 mg. calcium. No change in balance occurred on hydrocortisone or after adrenalectomy.

hypercalcemia, and hypophosphatemia returned At autopsy, a single bone metastasis was present. Recent analysis of the parathyroids in this case reveals a secondary hyperplasia.[7] Secondary hyperplasia of parathyroids also has been found in other cases of neoplasia with hypercalcemia with and without bone metastases. In such cases it seems reasonable to postulate that the cancer cells elaborate a substance that stimulates the parathyroid gland to cause rapid bone dissolution and resultant hypercalcemia.

RENAL EXCRETION OF CALCIUM

Endogenous calcium excretion in man, expressed as a ratio of urine excretion to fecal excretion, is approximately 2 to 1.[13] A similar excretion ratio exists in the monkey, but

in the dog the ratio is reversed to 1 to 10 and in the rat 1 to 22.[27] Consideration of excretion ratios must be taken into account when metabolic experiments in the dog and the rat are translated to man. With persistent hypercalcemia and hypercalciuria in breast cancer, metabolic experiments reveal no defect in absorption. When calcium intake is increased from 150 mg. to 1.0 Gm., about 20 per cent of calcium in the intake is absorbed and retained, producing a less negative balance and very little or no increase in urinary calcium excretion (Fig. 3). In such patients the normal excretory ratios usually are present, with the kidney playing the major role. Pearson states that when the urinary excretion of calcium exceeds 500 mg. per 24-hr. period, hypercalcemia occurs.[51] This is generally true, but in occasional patients urinary calciums as high as 700 mg. per 24 hours have been found to be associated with normal serum calcium.[6] In most of these latter cases an osteoblastic reaction is present within the bone, so that bone production in the host may play a part in keeping the serum calcium at a normal level. In addition, changes in renal reabsorptive mechanisms and levels of ionized calcium also must be important in regulating the level of total serum calcium. The exact mechanism of calcium excretion by the kidney is not known, due primarily to difficulties in measuring ionized and protein-bound calcium. All evidence seems to indicate that the calcium ion is filtered and partially reabsorbed by the renal tubule.[54] Indirect evidence indicating that calcium probably is reabsorbed can be seen in Figure 4, which demonstrates the effect of Mercuhydrin on calcium excretion. This patient was on a constant calcium intake and was receiving Meticorten, 20 mg. daily, orally for breast cancer metastatic to lungs and bone. With each injection of 2.0 cc. of Mercuhydrin, the concentration of calcium in milligrams per cent rose, accompanied by a moderate to minimal diuresis with resultant increase in total calcium output. A similar change has occurred during administration of Mercuhydrin to four additional patients, two of whom had no bone disease, but all were receiving cortisone therapy for varying rea-

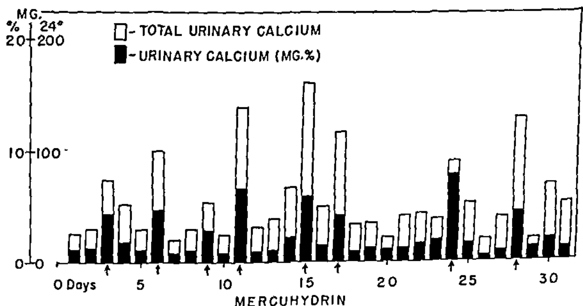


FIG. 4. The effect of Mercuhydrin in a 63-year-old woman with cancer of the breast metastatic to lungs and bones. The patient was on a constant low-calcium (150 mg.) diet and received 20.0 mg. of Meticorten orally. On eight different occasions, designated by the arrows, 2.0 cc. of Mercuhydrin was given intramuscularly, and each time the concentration of calcium rose with an increase in the total 24-hour urine output (Baker, W. H.: Am. J. Med. 21:716)

sons. Similar, although less marked, changes in normal subjects not on cortisone therapy have been reported elsewhere.[12]

During the acute phase of hypercalcemia, symptoms of dry mouth, polyuria, polydipsia and vomiting usually are present. We have studied two cases during this phase, and renal function studies have demonstrated a decreased glomerular filtration rate and a decreased renal blood flow with a normal filtration fraction. Both patients were unable to concentrate well but responded normally to antidiuretic hormone.[9] When calcium lactate infusions are given to normal subjects in amounts sufficient to cause hypercalcemia, increased urinary excretion of sodium and chloride accompanies increased calcium excretion.[44] The exact significance of these data is unknown but indicates a direct or indirect effect of calcium on kidney function.

With sustained hypercalcemia, deposition of calcium occurs within the renal tubules and in the pelvis with eventual nephrocalcinosis and at times stone formation, leading to mechanical impairment of kidney function with a rising nonprotein nitrogen and serum phosphorus. In such a situation hypercalcemia is further aggravated, and therapy must be directed toward its cause to interrupt a vicious cycle.

HYPERCALCEMIA

An elevated blood calcium can occur in many diverse pathologic states such as hyperparathyroidism, vitamin D intoxication, milk-alkali syndrome, Boeck's sarcoid and a wide variety of neoplastic diseases metastatic to bone.[3,18] It is not the purpose here to discuss the distinguishing features of each of these conditions, and it suffices to say that the conventional clinical history, physical examination and laboratory analysis usually are adequate to differentiate them.

Since the blood values of hyperparathyroidism and disseminated neoplasia, with or without bone disease, may be identical, the level of spinal fluid calcium has been advocated as a means of differentiation.[31] We have been unable to confirm this, and Figure 5 shows the level of spinal fluid calcium as

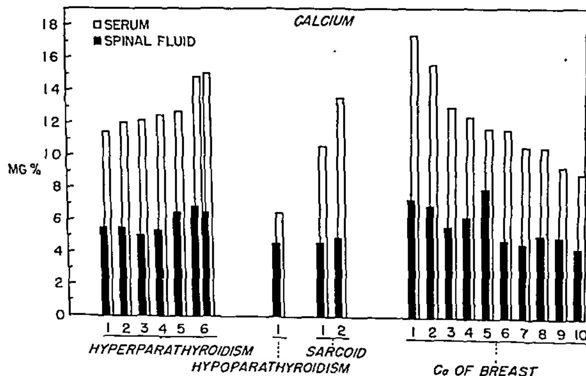


Fig. 5 Simultaneous serum and spinal fluid calciums. Note spinal fluid value has rough correlation with serum level and varies in narrow range of 4 to 7.8 mg. per cent. There is no difference between hyperparathyroidism and breast cancer.

compared with the levels of serum calcium in four different conditions. One can see that level of spinal fluid calcium varies directly with serum level within a narrow range.

Hypercalcemia occurring in the presence of neoplastic bone involvement has been well discussed by numerous authors.[1,2,29,38,57] It occurs spontaneously in about 10 per cent of cases with disseminated breast cancer and in about 20 per cent of breast cancers treated with hormones.[25] It occurs occasionally with hypernephroma, bronchogenic carcinoma, multiple myeloma and other miscellaneous tumors metastatic to bone, but it is very rare in thyroid carcinoma and absent in prostatic carcinoma. In prostatic carcinoma, the excess calcium released by bone breakdown may be compensated for by the very active osteoblastic proliferation of the host. On the other hand, in thyroid carcinoma with little osteoblastic proliferation, reason for its absence may be related to the very slow growth rate of thyroid cancer. As stated previously, hypercalcemia usually is associated with a normal or an elevated serum phosphorus. In the absence of liver or biliary tract disease, hypercalcemia usually, but not always, is accompanied by a normal or a falling serum alkaline phosphatase, and remission from hypercalcemia by a rising alkaline phosphatase, indicating bone repair.[25,37]

In our experience we have now seen two cases of hypercalcemia associated with cancer that have not shown evidence of bone metastases, either by roentgenogram or at autopsy.[14,15] Very recently two cases of bronchogenic carcinoma with hypercalcemia, hypercalciuria and hypophosphatemia, without evidence of bone disease, have been reported.[16] In one case, hypercalcemia subsided 4 days after removal of the primary lung tumor but recurred with metastases to the chest wall and then remitted prior to death. Routine autopsy showed no skeletal metastases, but areas of bone remote from the primary tumor showed active bone resorption without tumor cells. A second case showed no evidence of bone metastases by biopsy or roentgenogram, and with removal of the primary tumor hypercalcemia subsided An additional 10 cases of neoplasia with hypercalcemia and negative autopsy evidence of bone metastases have been reported by Plimpton and Gellhorn.[52] Three of these cases involved the lung and seven were of retroperitoneal origin, kidney, uterus and ovary. In three cases hypercalcemia subsided after removal of the primary tumor. It is very difficult to explain the above cases merely on the basis of tumor growth and osteolytic destruction of bone by tumor cells. As previously suggested by Albright,[3] it suggests that the tumor cells are elaborating some chemical which is capable of causing resolution of bone. The fact remains, however, that in many of these instances the tumors described have a high frequency of bone metastasis, and single small areas of metastatic lesions may be missed at routine autopsy. In addition, patchy necrosis is common in osteoclastic tumors, and at times this may be so extensive that tumor tissue is replaced by a structureless debris of an area several centimeters in diameter, so that the edge of the neoplasm may be devoid of living cells over a wide front.[59] Thus a small biopsy site may not include the neoplastic cells. The decrease in hypercalcemia following removal of the primary tumor is very good evidence that bone resorption is related definitely to the dividing neoplastic cells. Examination of the parathyroid glands in our two cases and in one of Connor's cases has revealed secondary parathyroid hyperplasia. Since hypercalcemia would suppress parathyroid activity, we believe, as stated previously, that the tumor cells may elaborate a substance to cause parathyroid hyperplasia and resultant increase in bone resorption

A few cases of metastatic bone cancer with hypercalcemia and low serum magnesium and increased urinary magnesium have been described recently. Convulsions oc-

curred in two, presumably due to the low serum magnesium. The ratio of magnesium to calcium in the urine was greater than could be accounted for solely as a result of dissolution of bone ($Mg^{++}/Ca^{++}=1/50$) and suggested that some of the magnesium came from a source other than bone.[20] Since similar changes in serum and urinary magnesium occur after administration of parathyroid extract and rarely in hyperparathyroidism, secondary parathyroid hyperplasia could be a reasonable explanation of the findings.

Since sudden death may occur in hypercalcemia, successful therapy may be a lifesaving maneuver. Numerous methods to lower the serum calcium with complexing and chelating agents have been tried. The administration of sodium citrate, a complexing agent, has been advocated frequently.[34] Very little lowering of the serum calcium occurs with sodium citrate, since the citrate is metabolized quickly. The attempt to lower the serum calcium by means of chelating agents such as sodium ethylenediaminetetraacetate (sodium EDTA) has not been successful.[19,30,55] Although a proportion of the serum calcium is bound and chelated by sodium EDTA and thence excreted in the urine, there is no change or an increase in the total serum calcium. Associated with the increase in serum calcium, an increase in urinary calcium occurs[19] Finally, sodium EDTA, when given in amounts to bind the excess ionized calcium, will produce renal tubular damage with anuria and azotemia to give further and sometimes permanent renal impairment.[19,23]

Use of oral agents such as sodium phytate or oral sodium EDTA to decrease intestinal absorption of calcium may be of temporary benefit.[6,28] However, from a practical standpoint, many of these patients are nauseated and vomiting actively, so that the use of oral agents is not feasible. The best available means to treat hypercalcemia in breast carcinoma at the present time is to administer large amounts of intravenous fluid to promote the renal excretion of calcium and prevent, by lowering of calcium concentration, nephrocalcinosis. When hypercalcemia follows administration of hormones, discontinuance of the offending hormone is indicated, as well as administration of large amounts of fluid. In spontaneous hypercalcemia, shifts in the hormonal environment by means of castration, adrenalectomy, hypophysectomy or administration of hormones may change the tumor-host relationship to favor the host, with subsequent decrease of the elevated blood calcium.

THE EFFECT OF HORMONES ON CALCIUM METABOLISM IN CANCER

In the final analysis, very little is known concerning the exact nature of action of hormones on calcium metabolism in man. Most of the information is derived from indirect parameters, and thus the changes that occur with administration or withdrawal of hormones must be analyzed with full awareness of the multiple known and unknown factors involved.

Aub, in 1933, reported four cases of breast cancer metastatic to bone which showed slight increases in urinary calcium that he interpreted to be due to the destructive effects of the tumor on bone. Parathyroid extract administered to these patients produced the same effect as in control subjects.[4]

ESTROGENS

Administration of estrogen to some patients with breast cancer metastatic to bone may be followed by a rapid appearance of hypercalcemia. This type of reaction usually occurs in 24 hours to 2 weeks after initiation of estrogen therapy and usually is accompanied by severe aching bone pain, nausea and vomiting, leading finally to stupor and coma. Such a patient is depicted in Figure 6. Discontinuance of estrogen in this case was followed by complete subsidence of hypercalcemia and an improvement in clini-

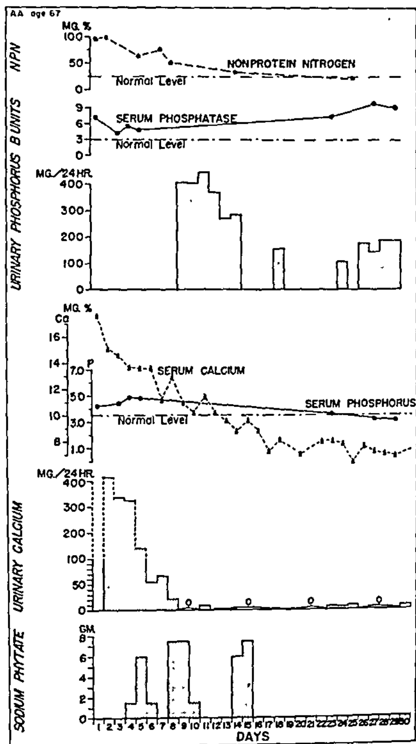


FIG. 6. Carcinoma of the breast with bone metastasis. Stilbestrol, 10.0 mg., was given postoperatively and was followed in 48 hours by development of hypercalcemia and hypercalciuria. Discontinuance of the hormone on the 1st day caused a complete remission of the hypercalcemia. Note slight increases in serum calcium after stopping Na phytate on the 7th and the 11th days.

cal condition with rising alkaline phosphatase and lowering of the urinary calcium Figure 7 represents an additional case of rapid onset of hypercalcemia following stilbestrol therapy. Continued administration of stilbestrol was accompanied by complete subsidence of hypercalcemia and radiologic evidence of recalcification of bone lesions in 4 months. Since this situation of hypercalcemia induced by hormones can be reversed by stopping the hormones or, in an occasional case, by continuation of hormones, it is obvious that some other factor besides estrogen is involved.

More recently, Pearson has shown by very careful metabolic studies that fluctuations in serum and urinary calciums in metastatic breast cancer can be correlated with hormonal changes during menstrual cycles and are indicative of hormone-dependent tumor

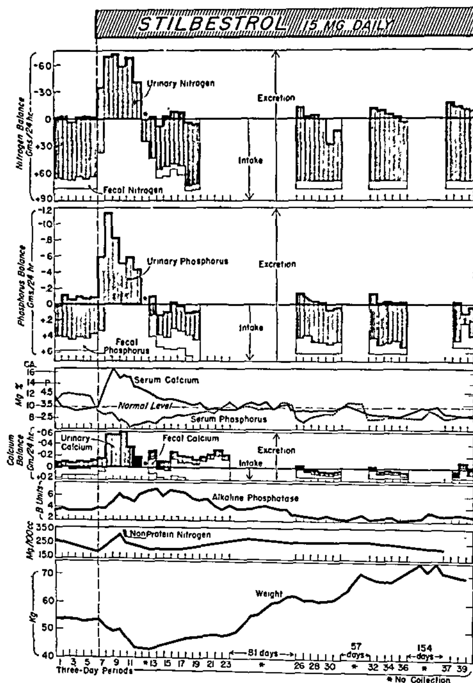


FIG. 7. Note the initial marked increase in serum and urinary calcium on stilbestrol, which later is followed by a decrease on continued administration, with eventual positive calcium balance and roentgenographic evidence of recalcification of lesions in 4 months. (Kennedy, B. J.: Cancer Res. 13:446)

cells.[51] If urinary calcium rises prior to the menses and falls following cessation of the menses, beneficial effect as measured by urinary calcium will occur after castration. On the basis of urinary calcium change, he postulates that there are two types of breast cancer: (1) those that are hormone dependent and are influenced adversely by estrogens, and (2) those that are hormone independent and are unaffected by estrogens.

Administration of estrogen also has been used in breast cancer with skeletal metastases in an attempt to select patients who will benefit from castration. If calcium excretion rises upon administration of estrogen, this indicates hormonal dependency and thus predicts those patients who will benefit from removal of estrogen sources. Emerson and Jessiman report a case of a 72-year-old female who obtained a gratifying objective response from castration after previous estrogen stimulation had caused a rise in urinary calcium.[21] In our experience, these methods for selecting patients for castration have

been helpful predominantly in the postmenopausal female. In the case of the estrogen stimulation test, we have not had 100 per cent correlation with the results predicted on the basis of calcium excretion. A few of our patients have had increases in urinary calcium excretion following estrogen administration and have shown no benefit whatsoever from castration. On the other hand, other patients who have had no increases in calcium excretion at the dose range of 5 mg. of stilbestrol daily have received definite benefit from castration, with recalcification of their bony lesions. In addition, we have encountered two patients, one of whom is depicted in Figure 7, who have demonstrated an initial increase in serum and urinary calcium but have received ultimate benefit from continued administration of stilbestrol. Thus the initial effect may not predict future bene-

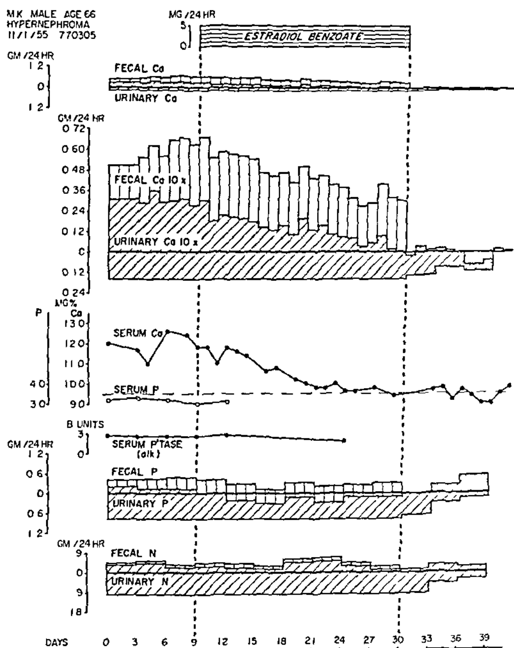


FIG. 8. Renal cell carcinoma with bone metastasis. Note gradual fall in serum and urinary calcium during treatment with estrogen. During this time roentgenograms demonstrated progression of lesions, and the patient died 14 days after the hormone was stopped.

fit in all cases. The exact effect of estrogens on bone metabolism in control subjects is not known, but in those patients who have postmenopausal osteoporosis a slight decrease in urinary calcium usually occurs, which in most cases is interpreted as repair of osteoporosis.[3] In such patients decreases in calcium excretion cannot be interpreted as complete evidence of beneficial antitumor effect. Estrogen may also decrease serum and urinary calcium in patients with metastatic bone tumors which are not considered to be hormone dependent (Fig. 8, [56]). Increases in calcium excretion following estrogen administration in mammary carcinoma are associated most probably with increases in growth rate of the tumor cells. In such cases inhibition of pituitary tropic hormones and/or growth hormones may be factors. However, it is equally possible that necrosis of tumor cells within the bone may occur similar to the necrosis which is produced in soft tissue and has been shown pathologically.[25]

ANDROGENS

Early observations by Adair and others that androgen administration in mammary carcinoma frequently may cause exacerbation of the disease, manifested by hypercalcemia and hypercalciuria, have been confirmed by many investigators.[1,29,46,54] Indeed, in spite of marked symptomatic relief obtained with androgen therapy, large increases in urinary calcium excretion and rapid dissolution of the bones can be seen by roentgenogram.[47,48] In most of the carefully studied cases in which androgen therapy has been continued, no reversal of hypercalcemia or hypercalciuria has occurred.[6,39] The cause of hypercalciuria and hypercalcemia, induced by androgens, is not known. Recent evidence has demonstrated the conversion of testosterone to estradiol by human tissue, and it is possible that such a conversion could cause an increased growth rate of estrogen-dependent cells with a resultant dissolution of bone.[5] However, this does not resolve the fact that in about 20 per cent of metastatic mammary cancers, androgens produce a beneficial recalcification of osteolytic lesions.[47,48] In some cases early increase in bone pain, nausea, vomiting and fever may predict a future beneficial effect of androgens, so that early symptomatic increase after administration of androgens may be only temporary and may indicate a future benefit in favor of the host and an osteoblastic reaction.

CORTISONE

The beneficial effects of cortisone in breast carcinoma have been well documented by many authors.[41,50,58] In many such cases cortisone has produced a lowering of the serum and urinary calciums. In a very few of these cases recalcification of the bones has occurred. We are unable to explain completely this effect of cortisone. Cortisone may act by inhibition of ACTH, with a resultant decreased output of sex steroids by the adrenal gland and thus decreased growth rate of estrogen-dependent mammary carcinoma cells. However, many other unknown factors may be involved.

ABLATIVE THERAPY

Castration and adrenalectomy may exert their beneficial effects upon calcium metabolism in neoplastic disease by removal of major estrogen sources which are stimulating the tumor cells, thus tilting the dynamic tumor-host relationship in favor of the host. Hypophysectomy may benefit by removing trophic stimulation of the adrenals and the ovaries, with resultant decrease in estrogen levels. In addition to the removal of gonadotropins, hypophysectomy removes thyrotropin and somatotropin. Both these latter hormones may cause increased urinary calcium excretion; thus, to judge the effect of hypophysectomy by decrease in urinary calcium may be hazardous In two patients, one reported by Pearson and the other by Jessiman, administration of growth hormone to hypophysectomized breast-cancer patients resulted in increased calcium excretion.[36,49]

Administration of human-growth hormone to a pituitary dwarf resulted in an increased calcium excretion in urine but a greater decrease in fecal excretion, producing a net positive calcium balance.[11] In addition, acromegaly is associated with an increase in urinary calcium,[10] so that the exact significance of increased calcium excretion following administration of growth hormone to cancer patients cannot be determined.

SUMMARY AND CONCLUSIONS

The various factors governing the presence of metastatic cancer in bone have been discussed. The reasons for this high incidence seem to be related to the vasculature and the fertile soil afforded by the skeletal system. Reasons for the various types of host reactions in bone are not fully explained and probably are related to the growth rate of the tumor cells and their individual biochemical characteristics. The dynamic equilibrium that exists between bone dissolution and bone production in metastatic mammary carcinoma is subject to spontaneous changes, at times in favor of the tumor cells and at times in favor of the host. It appears likely that hypercalcemia and/or hypercalciuria represent a change in favor of the tumor cells, usually accompanied by increased growth rate of tumor cells with a resultant destruction of bone and possibly of the tumor cells themselves. Secondary parathyroid hyperplasia can exist in the presence of hypercalcemia and neoplasia with or without bone metastasis and may be the cause of the hypercalcemia in such cases. The use of calcium excretion as a guide to future therapy is a gross but not unequivocal prediction of future benefit. The multiplicity of factors, both known and unknown, governing calcium excretion makes its use as a sole measure of objective effect unwise, and other parameters of antitumor effect should be used in conjunction. Recent reports on bone resorption effects associated with bronchogenic carcinoma and other tumors may lead eventually to a better understanding of osteolytic reactions produced by tumors remote from bone.

REFERENCES

1. *Adair, F. E.:* The use of the male sex hormone in women with breast cancer, Surg., Gynec. & Obst. 84:719, 1947.
2. *Adair, F. E., and Herrmann, J. B.:* The use of testosterone propionate in the treatment of advanced cancer of the breast, Ann. Surg. 123:1023, 1946.
3. Albright, F., and Reifenstein, E. C.: The Parathyroid Gland and Metabolic Bone Disease, Baltimore, Williams & Wilkins, 1948.
4. Aub, J. C., and Smithwick, R. H.: Lead treatment of cancer, New England J. Med. 208:310, 1933.
5. Baggett, B., Engel, L. L., Savard, K., and Dorfman, R. I.: Formation of estradiol-17β —C^{14} from testosterone-3 —C^{14}, by surviving human ovarian slices, Fed. Proc. 14: 1, 1955.
6. *Baker, W. H.:* Unpublished data.
7. Baker, W. H., and Castleman, B. J.: Unpublished data.
8. Baker, W. H., Stanbury, J. B., and Dow, E. C.: The use of radioactive calcium in the study of metastatic bone cancer, Proc Am. A. Cancer Res. 2:186, 1957.
9. Batson, O. V.: The function of the vertebral veins and their role in the spread of metastases, Ann. Surg. 112:138, 1940.
10. Bauer, W., and Aub, J. C.: Studies of calcium and phosphorus metabolism. XVI. The influence of the pituitary gland. J. Clin Invest 20:295, 1941.
11. Beck, J. C., McGarry, E. E., Dyrenfurth, I., and Venning, E. H.: The metabolic effects of human and monkey growth hormone in man, Program of the forty-ninth annual meeting of Am Soc. Clin. Invest., p. 9, May, 1957.
12. Blumgart, H. L., Gilligan, D. R., Levy, R. C., Brown, M. G., and Volk, M. C.: Action of diuretic drugs. I. Action of diuretics in normal persons Arch Int. Med. 54:40, 1934.
13. Bronner, F., Harris, R. S., Maletskos, C. J., and Benda, C. E.: Studies in calcium metabolism; the fate of intravenously injected radiocalcium in human beings, J. Clin. Invest. 35:78, 1956.
14. Case Records of the Massachusetts General Hospital: New England J. Med. 248:248, 1953.

15. ———: New England J. Med. 256:750, 1957.
16. Connor, T. B., Thomas, W. C., Jr., and Howard, J. E.: The etiology of hypercalcemia associated with lung carcinoma, J. Clin. Invest. 35:697, 1956.
17. Dent, C. E.: In Ciba Foundation Symposium on the Kidney, p. 242, Boston, Little, 1954.
18. Dent, C. E., Flynn, F. V., and Nabarro, J. D. N.: Hypercalcemia and impairment of renal function in generalized sarcoidosis, Brit. M. J. 2:808, 1953.
19. Dudley, H. R., Ritchie, A. C., Schilling, A., and Baker, W. H.: Pathologic changes associated with the use of sodium ethylene diamine tetra-acetate in the treatment of hypercalcemia, New England J. Med. 252: 331, 1955.
20. Eliel, L. P., Smith, W. O., and Alvarez, A. S.: The pathogenesis and management of metabolic disturbances in osteolytic bone disease, Proc. Am. A. Cancer Res. 2:198, 1957.
21. Emerson, K, Jr., and Jessiman, A. B.: Hormonal influences on the growth and progression of cancer, New England J. Med. 254:252, 1956.
22. Emerson, W. J., Kennedy, B. J., Graham, J. N., and Nathanson, I. T.: Pathology of primary and recurrent carcinoma of the human breast after administration of steroid hormones, Cancer 6:641, 1953.
23. Foreman, H., Finnegan, C., and Lushbaugh, C. C.: Nephrotoxic hazard from uncontrolled edathamil calcium-disodium therapy, J.A M A. 160:1042, 1956.
24. Freid, J. R., and Goldberg, H.: Treatment of metastases from cancer of the breast, Am. J. Roentgenol. 63:312, 1950.
25. Griboff, S. I, Herrmann, J. B., Smelin, A., and Moss, J.: Hypercalcemia secondary to bone metastases from carcinoma of the breast. I. Relationship between serum calcium and alkaline phosphatase values. J. Clin. Endocrinol. 14:378, 1954.
26. Gutman, E. B., Sproul, E. E, and Gutman, A. B: Significance of increased phosphatase activity at the site of osteoplastic metastases secondary to carcinoma of the prostate gland, Am. J. Cancer Res. 28:485, 1936.
27. Harris, R. S., *et al.*: Species differences in excretion of intravenously injected calcium[45], Fed. Proc. 15:2, 1956.
28. Henneman, P. H., and Carroll, E.: Suppression of urinary calcium and magnesium excretion by oral sodium phytate, J. Clin. Invest. 34:939, 1953.
29. Herrmann, J. B., Kirsten, E., and Krakauer, J. S.: Hypercalcemic syndromes associated with androgenic and estrogenic therapy, J. Clin. Endocrinol. 9:1, 1949.
30. Holland, J. F., Danielson, E., and Sahagian-Edwards, A.: Use of ethylene diamine tetra-acetic acid in hypercalcemic patients, Proc. Soc. Exper. Biol. & Med. 84:359, 1953.
31. Howard, J. E., Carey, R. A., Rubin, P. S, and Levine, M. D.: Diagnostic problems in patients with hypercalcemia, Tr. A. Am. Physicians 62:264, 1949.
32. Hyman, G. A., Elliott, R. H. E., Jr., and Sawitsky, A.: Studies on anemia of disseminated malignant neoplastic disease; hemolytic factor, Blood 9:911, 1954.
33. Hyman, G. A., and Harvey, J. L.: A comparison of bone-marrow aspiration and skeletal roentgenograms in the diagnosis of metastatic carcinoma, Cancer 8:576, 1955.
34. Hyman, G. A., Harvey, J. E., and Geldner, J. E.: The pathogenesis of anemia in patients with carcinoma, Am. J. Med. 19:350, 1955.
35. Jaimet, C. H., and Amy, H. E.: Cancer diagnosis by bone marrow smears, Ann. Int. Med. 44:617, 1956.
36. Jessiman, A. G., *in* Pearson, O. H. (Ed.): Hypophysectomy, p. 100, Springfield, Ill., Thomas, 1957.
37. Kennedy, B. J., Nathanson, I. T., Tibbetts, D. M., and Aub, J. C.: Biochemical alterations during steroid hormone therapy of advanced breast cancer, Am. J. Med. 19: 337, 1955.
38. Kennedy, B. J., Tibbetts, D. M., Nathanson, I. T., and Aub, J. C.: Hypercalcemia, a complication of hormone therapy of advanced breast cancer, Cancer Res. 13:445, 1953.
39. Laszlo, D., Schilling, A., Bellin, J., Gottesman, E. D., and Schulman, C. A.: Effect of testosterone on patients with bone metastases: a metabolic study, particularly of breast carcinoma, J.A.M.A. 148:1502, 1952.
40. Laszlo, D., Schulman, C. A., Bellin, J., Gottesman, E. D., and Schilling, A.: Mineral and protein metabolism in osteolytic metastases, J.A.M.A. 148:1027, 1952.
41. Lemon, H. M.: Cortisone-thyroid therapy of metastatic mammary cancer, Ann. Int. Med. 46:457, 1957.
42. Lemon, H. M., Davidson, M. M., and

Asimov, I.: Acid-phosphatase activity of normal and neoplastic human tissues, Cancer 7:92, 1954.
43. Lemon, H. M., and Reynolds, M. D.: Regression of mammary cancer metastases following decline of serum acid phosphatase induced by anti-estrogen therapy, Proc. Am. A. Cancer Res. 2:124, 1956.
44. Levitt, M. F., Halpern, M. H., Sweet, A. Y., and Gribetz, D.: The effect of intravenous calcium infusion on electrolyte excretion in man, J. Clin. Invest. 35:721, 1956.
45. Milch, R. A., and Changus, G. W.: Response of bone to tumor invasion, Cancer 9:340, 1956.
46. Myers, W. P. L., West, C. D., Pearson, O. H., and Karnofsky, D. A.: Androgen-induced exacerbation of breast cancer measured by calcium excretion, J.A.M.A. 161:127, 1956.
47. Nathanson, I. T.: Clinical investigative experience with steroid hormones in breast cancer, Cancer 5:754, 1952.
48. Nathanson, I. T., and Kelley, R. M.: Medical progress, hormonal treatment of cancer, New England J. Med. 246:135, 180, 1952.
49. Pearson, O. H. (Ed.): Hypophysectomy, p. 100, Springfield, Ill., Thomas, 1957.
50. Pearson, O. H., Li, M. C., MacLean, J. P., Lipsett, M. B., and West, C. D.: The use of hydrocortisone in cancer, Ann. New York Acad. Sc. 61:393, 1955.
51. Pearson, O H., West, C. D., Hollander, V. P., and Treves, N. E.: Evaluation of endocrine therapy for advanced breast cancer, J.A.M.A. 154:234, 1954.
52. Plimpton, C. H., and Gellhorn, A.: Hypercalcemia in malignant disease without evidence of bone destruction, Am. J. Med. 21:750, 1956.
53. Recklinghausen, von, F. D.: Die fibrose oder deformirende Ostitis, die Osteomalacie und die osteoplastische Carcinose in ihren gegenseitigen Beziehungen, pp. 1-89, pl 1-5, Berlin, 1891. Dedicated to Rudolf Virchow in celebration of his 71st birthday.
54. Smith, H. A.: Principles of Renal Physiology, p. 105, New York, Oxford, 1956.
55. Spencer, H., Greenberg, J., Berger, E., Perrone, M., and Laszlo, D.: Studies on the effect of ethylenediaminetetra-acetic acid in hypercalcemia, J. Lab. & Clin. Med. 47:29, 1956.
56. Spencer, H., Samachson, J., and Laszlo, D.: Clinical and metabolic studies of mytatrienediol, Proc. Am A. Cancer Res. 2:252, 1957.
57. Swyer, A. J., Berger, J. S, Gordon, H. M., and Laszlo, D.: Hypercalcemia in osteolytic metastatic cancer of the breast, Am. J. Med 8:724, 1950.
58 West, C. D., Li, M. C., MacLean, J. P., Escher, G. C., and Pearson, O. H.: Cortisone-induced remissions in women with metastatic mammary cancer, Proc. Am. A. Cancer Res 1:51, 1954.
59. Willis, R A.: The Spread of Tumours in the Human Body, ed 2, London, Butterworth, 1952.

Metabolismo de Calcium in Relation a Malignitate Metastatic

Summario in Interlingua

Le varie factores governante le presentia de cancere metastatic in osso es discutite. Le rationes pro iste alte incidentia es apparentemente relationate al vasculatura e al climate favorabile representate per le systema skeletic. Le rationes pro le varie typos de reaction in le ambiente ossee non es completemente clar. Illos es probabilemente connectite con le intensitate del crescentia del cellulos tumoral e con lor characteristicas biochimic individual. Le equilibrio dynamic que existe inter le dissolution e le production de osso in metastatic carcinoma mammari es characterisate per alterationes spontanee, a vices in favor del cellulas tumoral e a vices in favor del tessuto ambiente. Il es probabile que hypercalcemia e/o hypercalcuria representa un alteration in favor del cellulas tumoral, usualmente accompaniate de un crescentia accelerate del cellulas tumoral con resultante destruction de osso e possibilemente del cellulas tumoral mesme. Secundari hyperplasia parathyroide pote exister in le presentia de hypercalcemia e neoplasia

con o sin metastase ossee e es possibilemente le causa del hypercalcemia in tal casos. Le determination del excretion de calcium pote esser usate como guida in planar le therapia futur, sed como base del prediction de beneficios futur illo es grossier e non inequivoc. Le multiplicitate del factores—tanto cognoscite como etiam non cognoscite—que governa le excretion de calcium argue contra su uso exclusive como mesura de effectos objective. Altere mesurationes del effecto anti-tumoral debe esser utilisate in conjunction con illo. Recente reportos in re effectos resorptional in le osso in association con carcinoma bronchogene e altere tumores resultara forsan al futuro in un meliorate comprension del reactiones osteolytic producite per tumores de sito distante ab le osso.

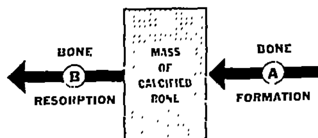
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The Relationships of Steroid Hormones to the Development and the Management of Osteoporosis in Aging People*

EDWARD C. REIFENSTEIN, JR., M.D.†

INTRODUCTION

All human tissues undergo changes with aging which we shall group together under the term *atrophy*. Bone is no exception, and the skeletal atrophy produces the metabolic bone condition which we call *osteoporosis*. This chapter deals with our present concepts of the relationships of steroid hormones (1) to the development of osteoporosis in aging people and (2) to the therapeutic management of this osseous disorder.



DYNAMIC EQUILIBRIUM

ARROW A = ARROW B
BOTH ARROWS = NORMAL

FIG. 1. Schematic diagram illustrating the dynamic processes affecting the bone mass of the normal adult man. This shows the mass of calcified bone in the normal adult in a state of dynamic equilibrium with the two separate metabolic processes, bone formation and bone resorption, taking place simultaneously at equal rates

OSTEOPOROSIS AND METABOLIC BONE DISORDERS

A short review of our interpretation of osteoporosis and certain related disorders will be presented in order to make clear exactly what is meant by these conditions.[8,94-96,98,100]

DYNAMIC PROCESSES AFFECTING BONE MASS

The mass of calcified bone in the body of an adult man in good health is in a state of dynamic equilibrium (Fig. 1), since two separate metabolic processes, bone formation and bone resorption, are taking place continuously, simultaneously and at such rates that the quantity of mineral that enters bone is equal to the quantity of mineral that leaves it. A change in the total mass of

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* Modified from an address entitled "Steroid Hormones and the Aging Skeleton" delivered by invitation at the annual meeting of the German Endocrinological Society, Freiburg im Bresgau, Germany, March 8, 1957.

† The Medical Division, The Squibb Institute for Medical Research, E. R. Squibb & Sons Division, Olin Mathieson Chemical Corporation, New York; and the Department of Medicine, New York Medical College, New York. The author wishes to thank Audrey Randall and her staff of secretaries and [illegible] to Don Forer and Paul Lawler for making charts

calcified bone can be brought about in two ways, (Fig. 2) either (1) by an alteration in the amount of bone formation or (2) by an alteration in the amount of bone resorption. Bone formation consists of two steps (Fig. 3): (1) the laying down of the protein matrix by the osteoblasts; and (2) the deposition of the calcium salts into this matrix. Because of these two steps, it is necessary to make a further division in the

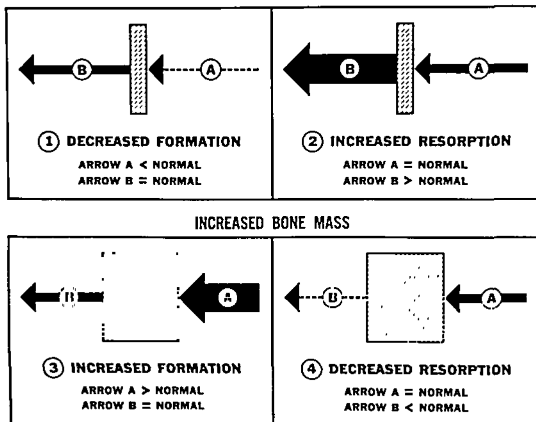


FIG. 2. Schematic diagrams illustrating the mechanisms for changing the total mass of calcified bone in the normal adult man. There are four divisions: (1) reduced calcified bone mass resulting from decreased formation; (2) reduced calcified bone mass resulting from increased resorption; (3) increased calcified bone mass resulting from increased formation; and (4) increased calcified bone mass resulting from decreased resorption.

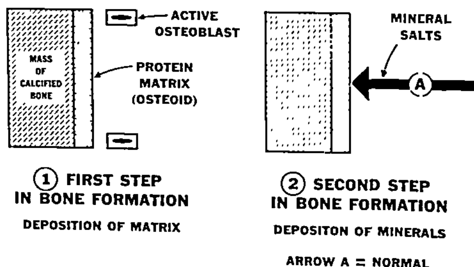


FIG. 3. Schematic diagrams illustrating the two steps of bone formation in the normal adult man. There are two divisions: (1) the first step in bone formation—the deposition of the protein matrix through the activity of the osteoblasts; and (2) the second step in bone formation—the deposition of the mineral salts into the matrix.

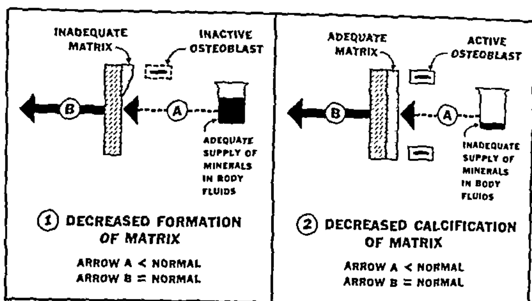


Fig. 4. Schematic diagrams illustrating the two mechanisms in man leading to decreased formation of calcified bone. There are two divisions: (1) decreased formation of calcified bone because of decreased formation of matrix, in which the osteoblasts are inactive although there is an adequate supply of minerals in the body fluids; and (2) decreased formation of calcified bone because of decreased calcification of matrix, in which the osteoblasts are active but there is an inadequate supply of minerals in the body fluids.

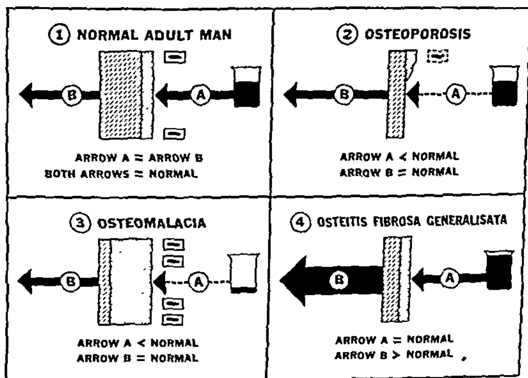


Fig. 5. Schematic diagrams illustrating the 3 mechanisms in man leading to "too little" calcified bone. There are four divisions. (1) the normal adult state with the mass of calcified bone in dynamic equilibrium; (2) the metabolic bone disorder, osteoporosis, with too little calcified bone mass because of too little formation of matrix, (3) the metabolic bone disorder, osteomalacia, with too little calcified bone mass because of too little calcification of matrix; and (4) the metabolic bone disorder, osteitis fibrosa generalisata, with too little calcified bone mass because of too much resorption of bone

the urine. The discrepancy between bone resorption and bone formation determines the degree of hypercalciuria. When the osteoporosis develops gradually, the increase in urinary calcium usually is not great; it may be marked when the condition develops rapidly. The reduced calcified mass of bone is indicated microscopically by fewer and less thick trabeculae rather than by a change in the bone contour, which is preserved until fracture occurs. The reduced skeletal mass is shown functionally by an increased susceptibility of the bone to fracture and compression.

Chronic (Clinical) Stage. As the bone mass steadily decreases, the skeleton becomes increasingly responsive to the stresses and strains of weight-bearing and reacts by applying a stronger and stronger stimulus to the sluggish osteoblasts. In time, the bone mass becomes so diminished that the skeleton responds to the stresses and strains with a sufficient increase in osteoblastic activity and bone formation so that (1) the bone mass, although reduced in quantity, again is restored to a dynamic equilibrium; (2) the serum alkaline phosphatase level, which is an index of osteoblastic activity, is within the physiologic range; (3) the osteoblasts and the osteoclasts appear qualitatively and quantitatively normal upon histologic examination; and (4) the hypercalciuria of the initial stage disappears.

The Diagnosis of Chronic (Clinical) Osteoporosis

There is no particular difficulty in diagnosing osteoporosis when skeletal deformities or clinical manifestations are present. We have pointed out (see Table 1) that too little calcified bone occurs in three conditions: osteoporosis, osteomalacia, osteitis fibrosa generalisata. It is impossible to differentiate these disorders merely from the evidence of a generalized reduced bone density in roentgenograms. Furthermore, all structural deformities resulting from insufficient bone strength can arise in any bone with a reduced calcified mass. Therefore, it is not surprising that the same anatomic defects (such as vertebral deformities) occur in all three disorders. Some differences in bone structure may be apparent to those experienced in interpreting roentgenograms.[79] Roentgenograms are of greater assistance in establishing the nature of bone disorder by indicating the location of the osseous lesions and the presence of other associated manifestations (such as bone cysts in osteitis fibrosa generalisata).

The Diagnosis of Early or Mild Osteoporosis

Cases Difficult to Diagnose. The major problem in diagnosing osteoporosis is in identifying the cases with early stages or mild states of generalized bone involvement. The decreased bone mass that exists prior to the development of structural deformities usually is not detectable at present because of the limitations in the technics currently available for studying human bones during life.

Diagnostic Value of Laboratory Procedures. Osteoporosis is not recognizable by clinical or laboratory procedures for several years. The concentrations of minerals in the body fluids are normal, even in the presence of marked osseous involvement. For this reason, serum determinations are of no diagnostic value. The ability of the skeleton to take up intravenously infused stable or radioactive calcium[72] or radioactive strontium[132] has been proposed as a method for detecting osteoporosis; these procedures may be valuable but need further study. Since the various areas of the skeleton differ markedly in anatomic structure, chemical composition and responsiveness to physiologic and pathologic influences, a piece of bone removed by biopsy is not a representative sample of the entire skeletal mass or of the bone from which the specimen was taken. Hence, data obtained from the study of bone samples may not indicate the true nature or degree of the generalized involvement. The technics for measuring mineral and nitrogen balances are expensive, time

TABLE 2. MINIMUM AMOUNT AND DURATION OF CALCIUM LOSS OR GAIN THAT CAN BE DETECTED BY VISUAL EXAMINATION OF ROENTGENOGRAMS IN CHRONIC (CLINICAL) OSTEOPOROSIS

AVERAGE NORMAL SKELETON	
Total calcium content	at least 1,150 Gm.
Minimum loss detectable by roentgenogram (from studies)	
Per cent of total calcium content	at least 30%
Amount of calcium	at least 345 Gm.
Minimum gain detectable by roentgenogram (assumed)	
Per cent of total calcium content	at least 30%
Amount of calcium	at least 345 Gm.
CHRONIC (CLINICAL) OSTEOPOROSIS—UNTREATED	
Average daily loss of calcium in balance studies	0.1-0.2 Gm.
Average time required to lose 345 Gm.	1,725-3,450 days
Minimum time for detectable loss by roentgenogram	4.7-9.7 yrs.
CHRONIC (CLINICAL) OSTEOPOROSIS—TREATED WITH STEROIDS	
Average daily gain of calcium in balance studies	0.1-0.2 Gm.
Average time required to gain 345 Gm	1,725-3,450 days
Minimum time for detectable gain by roentgenogram	4.7-9.7 yrs.

consuming and not suitable for routine evaluation of patients. The early stages or mild degrees of osteoporosis are not recognized in roentgenograms, because it is not possible to recognize changes in bone density with certainty until a considerable quantity of the calcium content has been lost. Most investigators[12,42,71] agree that it requires a minimum calcium loss of 30 per cent, and possibly up to 50 or 60 per cent, before a decrease in the calcified mass of bone can be recognized roentgenographically.[70]

Amount and Duration of Calcium Loss. Some calculations in Table 2 emphasize this difficulty. The total skeleton of the average normal man contains about 1,150 Gm. of calcium[106] Thirty per cent of this is 345 Gm. The average calcium loss by patients with untreated clinical osteoporosis during *metabolic balance studies* is about 100 to 200 mg. per day.[141] Therefore, the average time required to lose 345 Gm. of calcium would be from 1,725 to 3,450 days, or from 5 to 10 years![54,87] It should be noted that it is equally, if not more, difficult to detect repletion of the calcified bone mass by roentgenogram because, again, a considerable amount of calcium must be regained before an increase in bone density can be recognized.[26,54,87]

SENILE OSTEOPOROSIS

With these concepts as background, the osteoporosis of old age now will be considered. We refer to the metabolic bone disorder that occurs in persons over 45 years of age. In earlier reports[10,96] we divided the elderly women with clinical osteoporosis arbitrarily into two groups: (1) the postmenopausal cases, in which the clinical manifestations appeared before the age of 65; and (2) the senile cases, in which the clinical condition developed later. However, for this discussion we shall speak of the osteoporosis of old age as *senile osteoporosis* and include *postmenopausal osteoporosis* as a special type of senile osteoporosis.

RELATIONSHIPS OF STEROID HORMONES TO SENILE OSTEOPOROSIS

In 1940 Albright called attention to the frequent occurrence of osteoporosis in women about 5 to 10 years after the menopause and suggested that this bone disorder was another manifestation of the decreased ovarian hormone production that accompanied the climacteric state.[4] Subsequently, his clinic and associates published additional reports supporting and extending this

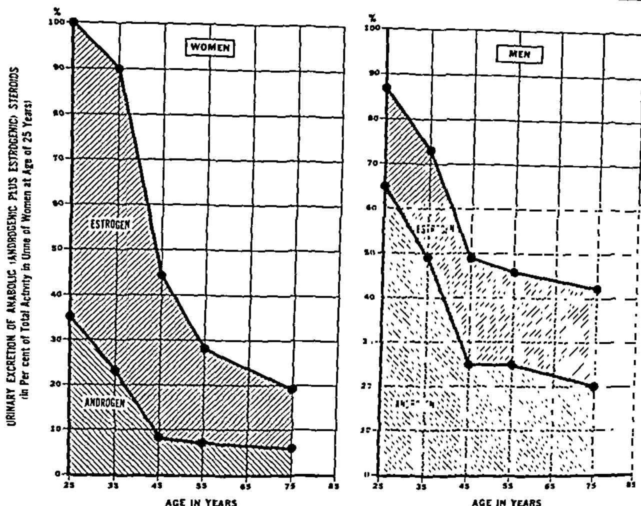


FIG. 6. Graphs illustrating the effect of age and sex upon the urinary excretion of steroids with anabolic activity (androgen plus estrogen).

The charts are derived from the data of Pincus, Dorfman and associates.[90-92] The graphs were constructed after the following steps (see Table 3): (1) the urinary estrogenic activity excreted at various ages was calculated for each sex in percentage of the amount excreted by women at the age of 25 years, (2) the urinary androgenic activity excreted at various ages was calculated for each sex in percentage of the amount excreted by men at the age of 25 years, (3) then for each sex the percentage excretions thus derived were added together at the respective age periods; (4) the resulting total excretion of androgenic and estrogenic activities was recalculated for each sex in percentage of the total quantity of steroids with these activities excreted by women at the age of 25 years; and (5) the resulting values were charted. The combined excretion of androgenic and estrogenic activities is assumed to be an index, if not a measure, of the production of anabolic steroids.

The graphs show that with declining years (1) the total production of anabolic steroids diminishes in both sexes and (2) the decreased production occurs at an earlier age, is more precipitous and reaches a considerably lower level in women than in men. For further discussion see text.

concept [1-5,8,10,16,46,47,54,63,67,78,79,94 96,98,100] The major relationships of steroid hormones to senile osteoporosis will be presented by discussing two questions (1) Is there a deficiency of anabolic steroid hormones in senile osteoporosis? (2) Is there an excess of antianabolic steroid hormones in this disorder?

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AGE RANGE (Yrs)	ANDROGEN* (Mg Androsterone Equivalents/1 Hr)	ESTROGEN* (Rat Units/ 24 Hrs)	ANDROGEN (% of Excretion of Men Aged 20-29)	ESTROGEN (% of Excretion of Women Aged 20-29)	TOTAL ANDROGEN PLUS ESTROGEN (Column A plus Column B)	ADJUSTED TOTAL (% of Column C Equivalent to % of Total Excretion of Women Aged 29-29)	ANDROGEN (% of Total in Column D)	ESTROGEN (% of Total in Column D)
WOMEN								
20-29	0.0817	33.62	53%	100%	153%	100%	35%	65%
30-39	0 0534	34.39	35%	102%	137%	90%	23%	67%
40-49	0 0193	18.75	12%	56%	68%	44%	8%	36%
50-59	0.0168	10.73	11%	32%	43%	28%	7%	21%
60-90	0.0126	6.96	8%	21%	29%	19%	6%	13%
MEN								
20-29	0.1546	11.50	100%	34%	134%	87%	65%	22%
30-39	0.1166	12.44	75%	37%	112%	73%	49%	24%
40-49	0.0607	12.01	39%	36%	75%	49%	25%	24%
50-59	0.0603	10.64	39%	32%	71%	46%	25%	21%
60-90	0.0484	10.95	31%	33%	64%	42%	20%	22%

* Data of Pincus, Dorfman and associates[90, 92]

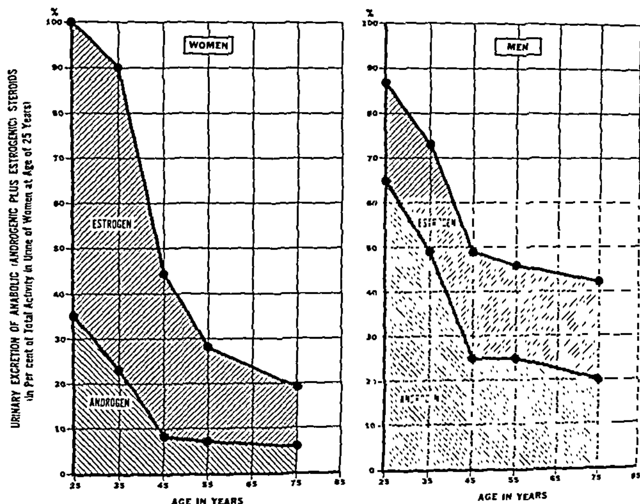


FIG. 6. Graphs illustrating the effect of age and sex upon the urinary excretion of steroids with anabolic activity (androgen plus estrogen).

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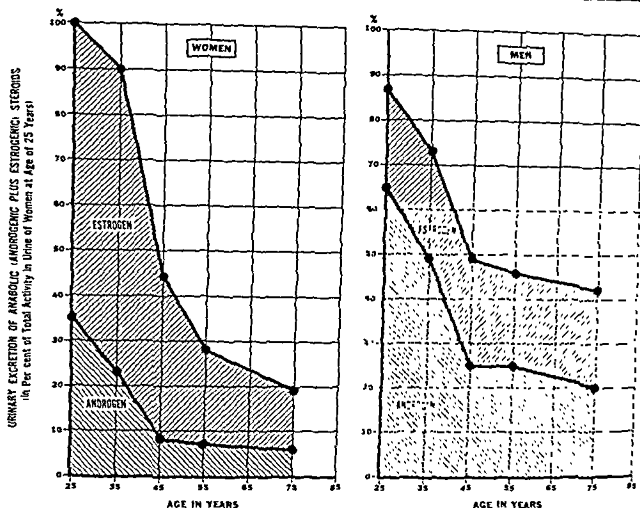


FIG. 6 Graphs illustrating the effect of age and sex upon the urinary excretion of steroids with anabolic activity (androgen plus estrogen).

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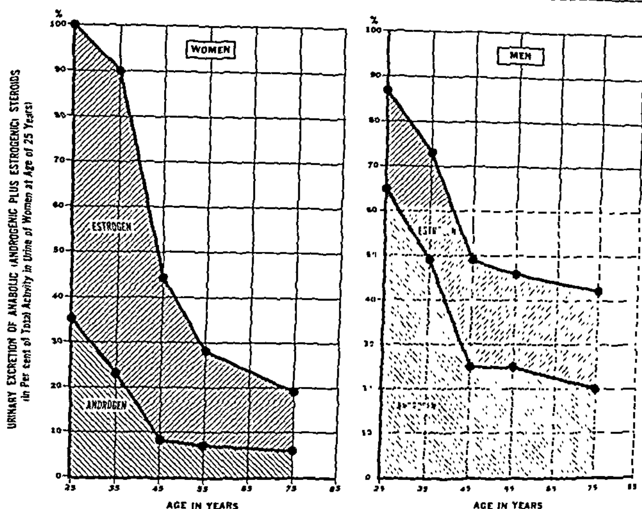


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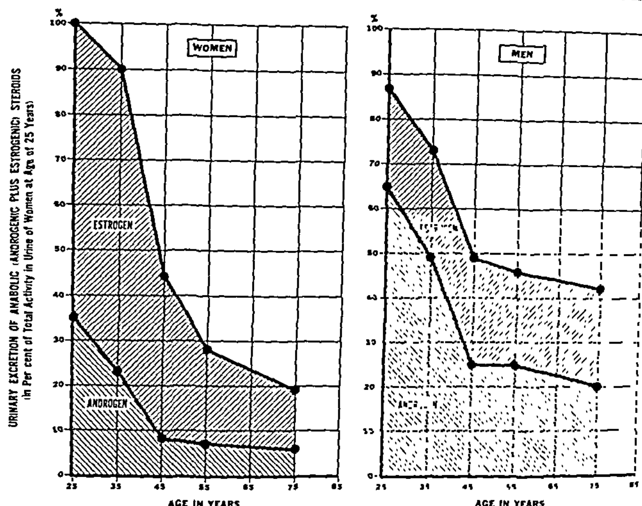


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The Nature of Anabolic Steroids

The androgenic and the estrogenic steroid hormones produced by the gonads and the adrenal cortex *stimulate the anabolism of*

effects are induced by androgen and estrogen, the term *androgenic* cannot be employed as a synonym for *anabolic.*

Primary Evidence Supporting Question 1

Anabolic Steroid Deficiency in Aging Persons. Some clinical features of aging (such as the reduction in muscle mass, the decrease in muscular power and activity, the increase in fatigability and the susceptibility of the bones to fracture) in themselves suggest a deficiency in anabolic steroids, since these manifestations are the opposite of those induced by these hormones.[97,125,143] The decrease with age in ovarian activity is clearly indicated by the cessation of menstruation; the decrease with age in testicular and adrenal cortical anabolic function is less easily recognized.

Recent studies on the urinary excretion of anabolic steroid metabolites provide more definitive evidence. These investigations establish that the production of androgen and of estrogen diminishes in both sexes with declining years.[41,51,52,90-92] The general pattern of these changes in women and in men at various ages is shown graphically in Figure 6, which has been prepared from data (Table 3) of Pincus, Dorfman and associates.[90-92] The combined excretion of androgenic and estrogenic activities is assumed to be an index, if not a measure, of the production of anabolic steroids. In women, the level of estrogen is high in the young adult and falls rapidly at the menopause to a value below that of men. The level of androgen in women also is high in the young adult (although below that of men) and, like that of estrogen, decreases markedly at the menopause. In men the level of androgen is high in the young adult and decreases more gradually with age, while the level of estrogen, which is low initially, shows little or no change throughout life.

The life history of the steroid anabolic hormones in the female is presented schematically in Figure 7. In this sex, steroids of the anabolic group are first produced in significant quantity at the time of puberty. Since anabolic steroids in the female have their origin both in the ovary and in the adrenal cortex, this means that there is not only a menarche with respect to the ovarian anabolic steroid hormones but also an adrenarche with respect to the anabolic steroid components.[3] The production of ovarian anabolic steroids falls rather precipitously at the menopause. The amount of anabolic steroids produced by the adrenal cortex also decreases with age but less abruptly; this decline has been termed an *adrenopause* with respect to these anabolic steroids.[3]

The life history of these steroids in the male is presented schematically in Figure 8. In this sex, steroids of the anabolic group likewise are first produced in a significant quantity at the time of puberty. Since both the testicular Leydig cells and the adrenal cortex contribute hormones with anabolic activity, the events of puberty involving these glands have been called a *leydigarche* and an *adrenarche* with respect to the anabolic ster-

TABLE 4. THE SEX DISTRIBUTION OF SENILE OSTEOPOROSIS

Investigators	Number of Men	Number of Women	Total Cases
Albright, Smith and Richardson[10]	2	37	39
Cooke[26]	7	43	50
Black, Ghormley and Camp[19]	41	167	208
Perloff, Boutwell and Maas[87]	12	50	62
Total cases	62	297	359
Per cent of total cases	17%	83%	100%

TABLE 5. THE AGE DISTRIBUTION OF THE ONSET OF SENILE OSTEOPOROSIS

INVESTIGATORS	AGE RANGE 30-39		AGE RANGE 40-49		AGE RANGE 50-59		AGE RANGE 60-90		TOTAL CASES ALL AGES
	Women	Men	Women	Men	Women	Men	Women	Men	
Albright, Smith and Richardson[10]	0	0	10	2	15	0	12	0	39
Cooke[26]	1	1	4	0	8	3	30	3	50
Perloff, Boutwell and Maas[87]	3	0	12	2	31	4	4	6	62
Total cases	4	1	26	4	54	7	46	9	151
Total women	4		26		54		46		130
Total men	1		4		7		9		21
Total women and men	5		30		61		55		151

AGE RANGE (Yrs)	WOMEN No. of Cases	WOMEN % of Total Cases	MEN No of Cases	MEN % of Total Cases	WOMEN AND MEN No of Cases	WOMEN AND MEN % of Total Cases
30-39	4	2.6%	1	0.7%	5	3.3%
40-49	26	17.2%	4	2 6%	30	19.8%
50-59	54	35.8%	7	4.6%	61	40 4%
60-90	46	30.5%	9	6 0%	55	36.5%
Total cases	130	86.1%	21	13.9%	151	100.0%

oids.[3] The production falls gradually in the aging male, approximately at the time of the female adrenopause. Thus, the male has an adrenopause and at the same time a leydigpause with respect to these hormones.[3]

Association in Aging Persons of Osteoporosis with Deficiency of Anabolic Steroids. Senile osteoporosis occurs much more frequently in aging women than in aging men.[10,19,26,87] In 359 cases of senile osteoporosis (Fig. 9 & Table 4), 83 per cent were women and 17 per cent were men; that is, there were five times as many cases in women as in men. Furthermore, women develop clinical manifestations of the bone disorder at an earlier age than men.[10,26,87] In 130 women with senile osteoporosis (Fig. 10 & Table 5), there was a rapid increase in the number of cases to the age of 55 and then a moderate decrease thereafter; while in 21 men there was a gradual steady rise in the number of cases as age advanced.[10,26,87]

Senile osteoporosis tends to be more severe in elderly women than in aging men. A survey of 136 ambulatory and apparently healthy persons, whose ages ranged from 63 to 95 years, revealed asymptomatic vertebral fractures by roentgenographic examination in 29 per cent of the 82 women and in only 20 per cent of the 54 men.[43] Postmenopausal women are more susceptible than men of comparable age to the harmful effects upon bone of immobilization[10] or of chronic corticoid therapy.[97] Removal of the ovaries accelerates the appearance of senile osteoporosis. In 10 women with artificial menopause, the average age at the onset of the osseous condition was 52 years, while in 27 women with physiologic menopause it was 56 years.[10]

These observations indicate that during the same period of aging two events take place: (1) the anabolic steroid hormone production decreases more abruptly and to much lower levels in women than in men and (2) senile osteoporosis develops more rapidly and with much greater frequency and severity in women than in men. The simi-

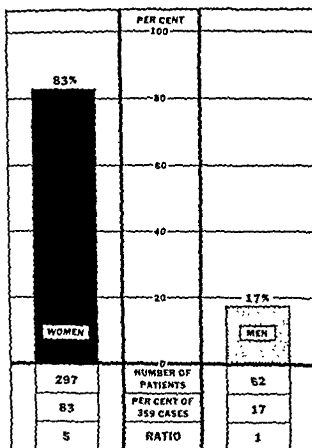


FIG. 9. The sex distribution of senile osteoporosis. This figure is based on observations of 359 cases of senile osteoporosis collected from the literature.[10,19,26,87] The data are given in Table 4. The average age of the patients was approximately 60 years. It will be seen that approximately 5 times as many women (83%) as men (17%) are affected. For further discussion see text.

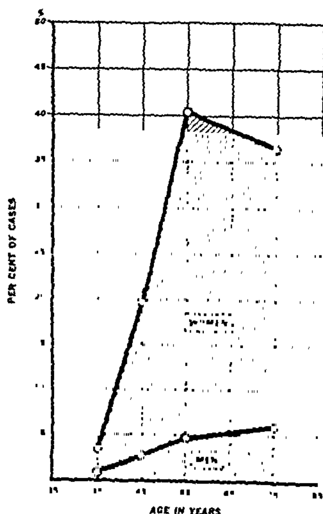


FIG. 10. The age distribution of the onset of senile osteoporosis. This is based on observations of 151 cases of senile osteoporosis collected from the literature.[10,26,87] The data are given in Table 5. It will be seen that in the 130 women there was a rapid increase in the number of cases to the age of 55 and a moderate decrease thereafter, while in the 21 men there was a gradual steady rise in the number of cases as age advanced. For further discussion see text.

larity in the time sequence and the course of these events suggests that they are related to each other.

The relationships are shown graphically in Figure 11, which is derived from the data in Figures 6 and 10. In women the rapid fall in anabolic steroid excretion to well below the 50 per cent baseline correlates in time with the rapid and marked rise in the percentage of cases of senile osteoporosis. In contrast, in men the more gradual fall in excretion to a level only slightly below the baseline correlates in time with the more gradual and less marked rise in the percentage of cases.

Effect of Anabolic Steroid Therapy on Osteoporosis in Aging Persons. In 1940 Albright made a preliminary report,[4,10] and in 1947 Reifenstein and Albright[98] published detailed observations extending over a period of 8 years on the favorable effects of androgen alone, of estrogen alone, and of these two anabolic steroids in combination, upon the clinical manifestations and metabolic balances of protein and osseous constituents in women and in men with senile

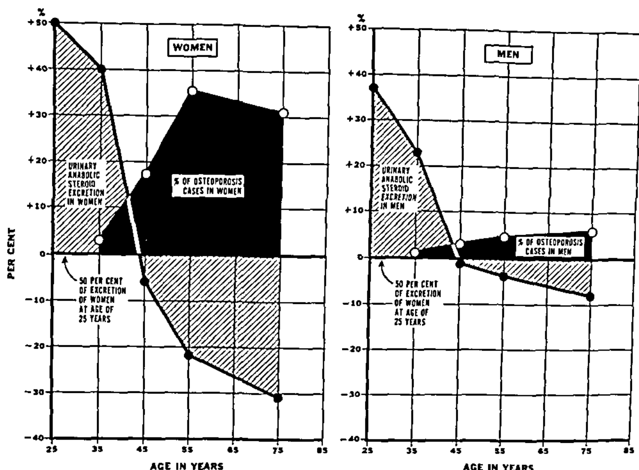


FIG. 11. The association in aging persons of senile osteoporosis with anabolic steroid deficiency.

The chart is derived from the data in Figures 6 and 10.[10,26,87,90-92] The calculations are given in the caption for Figure 6. However, in Figure 7 the baseline is constructed so that it represents 50 per cent of the maximal amount of anabolic (androgenic plus estrogenic) activity excreted by women at the age of 25 years. It will be seen that in women the rapid fall in anabolic steroids to well below the 50 per cent baseline correlates in time with the rapid and marked rise in the percentage of cases of senile osteoporosis. In contrast, in men the more gradual fall in excretion to a level only slightly below the baseline correlates in time with the more gradual and less marked rise in the percentage of cases. For further discussion see text.

osteoporosis. These reports have been amplified in later communications.[1-3,5,8,94 96,100]

We found that clinically the patients with senile osteoporosis responded very satisfactorily to anabolic steroid therapy. In the metabolic balance studies we evaluated relatively large doses of estradiol benzoate, diethylstilbestrol, testosterone propionate and 17-methyltestosterone; the minimum effective dosage was not determined. Our data demonstrated that estrogen alone had a greater effect on calcium retention than androgen alone, whereas androgen alone had a greater effect on nitrogen retention than estrogen alone; however, the combination of androgen and estrogen induced a greater retention of calcium than either steroid alone. Therefore, for therapy we recommend that these anabolic steroids should be combined

A portion of one metabolic balance study on a 72-year-old man with senile osteoporosis[8,98,99] is shown graphically in Figure 12. The chart has two divisions: (A) the measured nitrogen balance and (B) the measured nitrogen balance with superimposed theoretic nitrogen balance explainable by the measured phosphorus balance (after the phosphorus theoretically retained with cal-

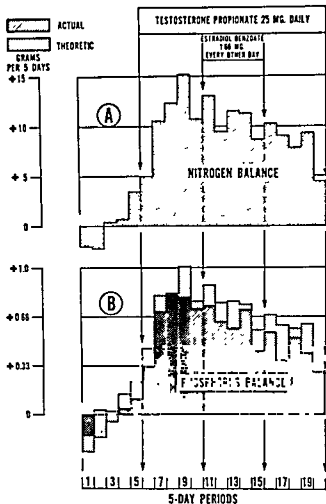


FIG. 12. The effect of anabolic steroid therapy upon the nitrogen and the phosphorus balances of a man with senile osteoporosis.

This metabolic balance study of a 72-year-old man with osteoporosis consists of 20 5-day periods. The scales for the balances in grams per 5-day period are given as the ordinates; the scale of the 5-day periods is given as the abscissa. The horizontal line starting at zero on each ordinate is the baseline of that particular balance; balances extending from the baselines toward the tops of the diagrams are positive; those extending from the baselines toward the bottoms of the diagrams are negative. The balances are charted as deviations from the average of the control periods rather than as the balances actually measured. The scales are chosen so that 1 unit of phosphorus is equal to 15 units of nitrogen to express the constant interrelationship of these substances that exists in normal protein tissues; thus the charted area of the phosphorus balance (after the phosphorus theoretically retained with calcium has been subtracted) should equal the charted area of the nitrogen balance when protein tissues have been retained. The factors for the calculations are given in Table 8 in Albright and Reifenstein;[5] the methods for the accumulation, the presentation and the interpretation of these data are discussed in detail in Reifenstein, Albright and Wells.[99] Testosterone propionate and estradiol benzoate were administered intramuscularly during the periods indicated.

The figure has two divisions: (A) the measured nitrogen balance; and (B) the measured nitrogen balance with superimposed theoretic nitrogen balance explainable by the measured phosphorus balance (after the phosphorus theoretically retained with calcium has been subtracted). It will be seen that there is a close correspondence between the measured and the theoretic nitrogen balances. This is evidence that testosterone propionate alone and in combination with estradiol benzoate induced a retention of nitrogen and phosphorus in the proportions that exist in muscle protoplasm. For an additional analysis on this patient see Figure 13.

(Data recharted from Case 6 in Reifenstein and Albright: J. Clin. Invest. 26:24-56. For further data on this patient see Albright;[1-3] Reifenstein, Albright and Wells;[99] and Case 13 (Figs. 78 & 155) in Albright and Reifenstein[5])

cium has been subtracted). There is a close correspondence between the measured and the theoretic nitrogen balances. This is evidence that testosterone propionate alone and in combination with estradiol benzoate induced a retention of nitrogen and phosphorus in the proportions that existed in protein tissues.

Another portion of this balance study[8,98,99] is given in Figure 13. This chart has two divisions: (A) the measured calcium balance and (B) the measured calcium balance with superimposed theoretic calcium balance explainable by the measured phosphorus balance (after the phosphorus theoretically retained with nitrogen has been subtracted). Again, there is a close correspondence between the measured and the theoretic calcium balances. This is evidence that testosterone propionate alone and in combination with estradiol benzoate induced a retention of calcium and phosphorus in the proportions that existed in bone.

Our observations on the effectiveness of

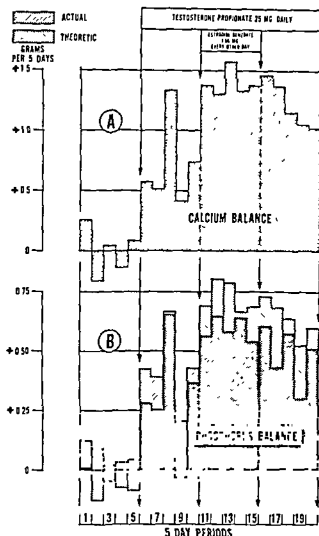


Fig. 13. The effect of anabolic steroid therapy upon the calcium and the phosphorus balances of a man with senile osteoporosis.

This metabolic balance study of a 72-year-old man with osteoporosis consists of 20 5-day periods. The method of charting these data is described in the caption for Figure 12. In this chart the scales are chosen so that 1 unit of phosphorus is equal to 2 units of calcium to express the constant interrelationship of these substances that exists in normal osseous tissues; thus the charted area of the phosphorus balance (after the phosphorus theoretically retained with nitrogen has been subtracted) should equal the charted area of the calcium balance when osseous tissues have been retained. The factors for the calculations are given in Table 8 in Albright and Reifenstein;[8] the methods for the accumulation, the presentation and the interpretation of these data are discussed in detail in Reifenstein, Albright and Wells.[99]

The figure has two divisions: (A) the measured calcium balance; and (B) the measured calcium balance with superimposed theoretic calcium balance explainable by the measured phosphorus balance (after the phosphorus theoretically retained with nitrogen has been subtracted). It will be seen that there is a close correspondence between the measured and the theoretic calcium balance. This is evidence that testosterone propionate alone and in combination with estradiol benzoate induced a retention of calcium and phosphorus in the proportions that exist in bone. For an additional analysis on this patient see Figure 12. For further discussion see text.

(Data recharted from Case 6 in Reifenstein & Albright: J. Clin. Invest. 26:24-56. For further data on this patient see Albright;[1-3] Reifenstein, Albright & Wells;[99] and Case 13 (Figs. 78 & 155) in Albright & Reifenstein[8])

anabolic steroids as therapy for senile osteoporosis have been confirmed by many investigators[11,13-15,21,26,45,46,50,53,54,69,73,74,76,77,86,87,104,105,107-112,120,137,139,141,144] who have evaluated the clinical response and occasionally, metabolic balance studies. By combining the published balance data that were reported in detail[21,111] with those from our own investigations,[98] we have calculated the average retention of minerals and nitrogen during the first 30 days of anabolic steroid therapy (Fig. 14 & Table 6). The dosage of anabolic steroids in these studies was: 25 to 50 mg. per day of testosterone propionate by injection; 375 mg. of free testosterone as pellets (evaluated during the first 21 days after implantation); 40 to 100 mg. per day of 17-methyltestosterone by mouth; 0.55 to 1.66 mg. per day of estradiol benzoate by injection; and 6 to 7 mg. (average) per day of diethylstilbestrol by mouth. The analysis shows that estrogen alone induces a greater retention of calcium and a lesser retention of nitrogen than androgen alone. Although the combination of both anabolic hormones does not cause a greater nitrogen retention, it results in a considerably greater calcium retention than either estrogen or androgen alone.

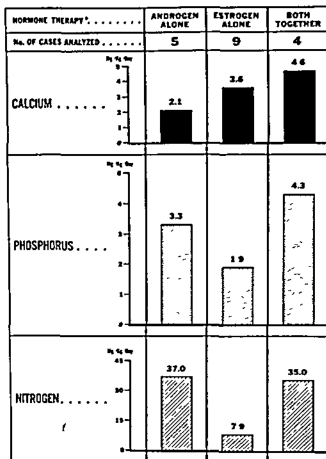


FIG. 14. Anabolic steroid therapy in senile osteoporosis: average retention of minerals and nitrogen during the first 30 days of administration of large doses.

The scales for the average net retention in mg. per Kg. per day are given as the ordinates; the results for 5 patients on androgen alone, for 9 patients on estrogen alone and for 4 patients on both hormones together are given on the abscissa. The periods of analysis during therapy ranged from 24 to 72 days. The data are given in Table 6. The scales are chosen so that 1 unit of phosphorus is equal to 2 units of calcium and 15 units of nitrogen to express the constant interrelationships of these substances that exist in normal protein and osseous tissues, thus the height of the phosphorus column should equal the sum of the columns for nitrogen and calcium when protein and osseous tissues have been retained. The factors for the calculations are given in Table 8 in Albright and Reifenstein;[8] the methods for the accumulation, the presentation and the interpretation of these data are discussed in detail in Reifenstein, Albright and Wells.[90]

It will be seen that, when estrogen is given alone, there is a greater retention of calcium and a lesser retention of nitrogen than when androgen is given alone. Note further that, while the combination of both gonadal hormones does not result in a significantly greater nitrogen retention than is obtained with androgen alone, the combination results in a greater calcium retention than with either estrogen or androgen alone. These findings are evidence that androgen and estrogen have qualitatively identical but quantitatively different somatotropic effects on protein and osseous tissues. For further discussion see text.

(Modified from Reifenstein, E. C., Jr.: Metabolic disorders of bone *in* Harrison, T. R. (Ed.): Principles of Internal Medicine, ed. 2, New York, Blakiston)

These findings are evidence that androgen and estrogen have qualitatively identical but quantitatively different anabolic effects upon protein and osseous tissues.

Some additional calculations from the data in Figure 14 are of interest. The theoretic phosphorus retention* has been calculated and compared with the measured phosphorus retention (Tables 6 & 7). The values agree very well. In fact, the correspondence is surprisingly good considering the difficulties in carrying out balance studies in man! This agreement provides strong evidence that in patients with senile osteoporosis the anabolic steroids induce a retention of nitrogen and phosphorus in the proportions that exist in normal protein tissue, and also a retention of calcium and of additional phosphorus in the proportions that exist in normal bone. Furthermore, androgen alone, estrogen alone, and both steroids in com-

* Th[illegible] (that is, the calcium retention divided by 2.3, the calcium to phosphorus ratio in normal bone) and the phosphorus that theoretically should have been retained with nitrogen (that is, the nitrogen retention divided by 15, the nitrogen to phosphorus ratio in normal muscle protein).

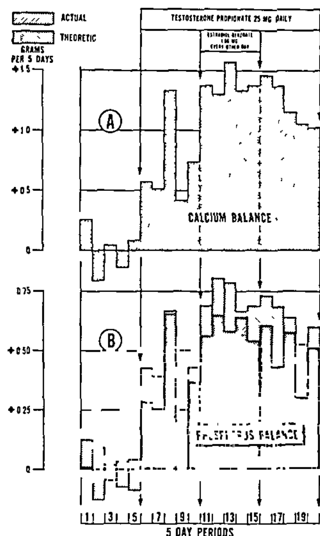


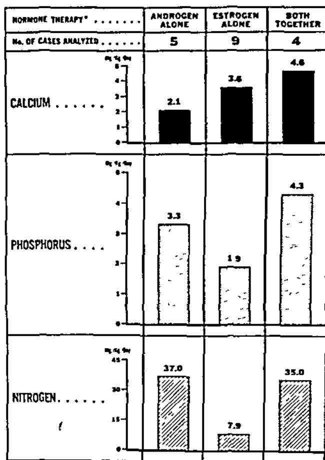
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This metabolic balance study of a 72-year-old man with osteoporosis consists of 20 5-day periods. The method of charting these data is described in the caption for Figure 12. In this chart the scales are chosen so that 1 unit of phosphorus is equal to 2 units of calcium to express the constant interrelationship of these substances that exists in normal osseous tissues; thus the charted area of the phosphorus balance (after the phosphorus theoretically retained with nitrogen has been subtracted) should equal the charted area of the calcium balance when osseous tissues have been retained. The factors for the calculations are given in Table 8 in Albright and Reifenstein;[8] the methods for the accumulation, the presentation and the interpretation of these data are discussed in detail in Reifenstein, Albright and Wells.[99]

The figure has two divisions: (A) the measured calcium balance; and (B) the measured calcium balance with superimposed theoretic calcium balance explainable by the measured phosphorus balance (after the phosphorus theoretically retained with nitrogen has been subtracted). It will be seen that there is a close correspondence between the measured and the theoretic calcium balance. This is evidence that testosterone propionate alone and in combination with estradiol benzoate induced a retention of calcium and phosphorus in the proportions that exist in bone. For an additional analysis on this patient see Figure 12. For further discussion see text.

(Data recharted from Case 6 in Reifenstein & Albright: J. Clin. Invest. 26:24-56. For further data on this patient see Albright;[1-3] Reifenstein, Albright & Wells,[99] and Case 13 (Figs. 78 & 155) in Albright & Reifenstein[8])

anabolic steroids as therapy for senile osteoporosis have been confirmed by many investigators[11,13 15,21,26,45,46,50,53,54,69,73,74,76,77,86,87,104,105,107 112,120,137,138,141,144] who have evaluated the clinical response and occasionally, metabolic balance studies. By combining the published balance data that were reported in detail[21 111] with those from our own investigations,[98] we have calculated the average retention of minerals and nitrogen during the first 30 days of anabolic steroid therapy (Fig. 14 & Table 6). The dosage of anabolic steroids in these studies was: 25 to 50 mg. per day of testosterone propionate by injection; 375 mg. of free testosterone as pellets (evaluated during the first 21 days after implantation); 40 to 100 mg. per day of 17-methyltestosterone by mouth; 0.55 to 1.66 mg. per day of estradiol benzoate by injection; and 6 to 7 mg. (average) per day of diethylstilbestrol by mouth. The analysis shows that estrogen alone induces a greater retention of calcium and a lesser retention of nitrogen than androgen alone. Although the combination of both anabolic hormones does not cause a greater nitrogen retention, it results in a considerably greater calcium retention than either estrogen or androgen alone.



*ANDROGENS Testosterone, Testosterone Propionate, 17 Methyltestosterone
ESTROGENS Estradiol Benzoate, Diethylstilbestrol

Fig. 14. Anabolic steroid therapy in senile osteoporosis: average retention of minerals and nitrogen during the first 30 days of administration of large doses.

The scales for the average net retention in mg. per Kg. per day are given as the ordinates; the results for 5 patients on androgen alone, for 9 patients on estrogen alone and for 4 patients on both hormones together are given on the abscissa. The periods of analysis during therapy ranged from 24 to 72 days. The data are given in Table 6. The scales are chosen so that 1 unit of phosphorus is equal to 2 units of calcium and 15 units of nitrogen to express the constant interrelationships of these substances that exist in normal protein and osseous tissues; thus the height of the phosphorus column should equal the sum of the columns for nitrogen and calcium when protein and osseous tissues have been retained. The factors for the calculations are given in Table 8 in Albright and Reifenstein;[8] the methods for the accumulation, the presentation and the interpretation of these data are discussed in detail in Reifenstein, Albright and Wells.[99]

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(Modified from Reifenstein, E. C., Jr.: Metabolic disorders of bone *in* Harrison, T. R. (Ed.): Principles of Internal Medicine, ed 2, New York, Blakiston)

These findings are evidence that androgen and estrogen have qualitatively identical but quantitatively different anabolic effects upon protein and osseous tissues.

Some additional calculations from the data in Figure 14 are of interest. The theoretic phosphorus retention* has been calculated and compared with the measured phosphorus *retention (Tables 6 & 7)*. The values agree very well. In fact, the correspondence is surprisingly good considering the difficulties in carrying out balance studies in man! This agreement provides strong evidence that in patients with senile osteoporosis the anabolic steroids induce a retention of nitrogen and phosphorus in the proportions that exist in normal protein tissue, and also a retention of calcium and of additional phosphorus in the proportions that exist in normal bone. Furthermore, androgen alone, estrogen alone, and both steroids in com-

* The theoretic ph[illegible] phosphorus ratio in normal bone) and the phosphorus that theoretically should have been retained with nitrogen (that is, the nitrogen retention divided by 15, the nitrogen to phosphorus ratio in normal muscle protein).

TABLE 6. ANABOLIC STEROID THERAPY IN SENILE OSTEOPOROSIS: MINERALS AND LARGE DOSES (AVERAGE

Sex of Patient	Investigator and Cases	Body Weight (Approx.) (Kg)	Days on Therapy	Calcium: Control (Mg./24 Hrs)	Calcium: Retention (Mg./24 Hrs.)	Calcium: Net Retention (Mg/24 Hrs)	Calcium: Net Retention (Mg/Kg/Day)
	Androgen Alone						
F	Reifenstein & Albright[98] Case 4a	48	24	− 146	+ 77*	+ 223	+4.6
F	Reifenstein & Albright[98] Case 4b	51	24	+ 148	+ 161*	+ 13	+0.25
M	Reifenstein & Albright[98] Case 6	70	20	+ 145	+ 295*	+ 150	+2.1
M	Shorr *et al.*[111] Case J. McD.	70†	28	+ 161	+ 241	+ 80	+1.1
F	Shorr *et al.*[111] Case M. O'H.	70†	21	+ 146	+ 321	+ 175	+2.5
	Average		23 4				+2.1
	Estrogen Alone						
F	Reifenstein & Albright[98] Case 1	51	20	− 65	+ 102*	+ 167	+3.3
F	Reifenstein & Albright[98] Case 2	54	35	− 21	+ 104*	+ 125	+2.3
F	Reifenstein & Albright[98] Case 3	74	40	0	+ 76*	+ 76	+1.0
F	Reifenstein & Albright[98] Case 5	37	48	− 305	+ 41*	+ 346	+9.3
M	Reifenstein & Albright[98] Case 6	67	20	+ 236	+ 296*	+ 60	+0.90
F	Shorr *et al.*[111] Case E. P.	70†	21	− 29	+ 309	+ 338	+4.8
M	Bogdonoff *et al.*[21] Case W. Ra.	87	15	+ 54	+ 149	+ 95	+1.1
M	Bogdonoff *et al*[21] Case G. S	59	15	+ 11	+ 167	+ 156	+2.6
M	Bogdonoff *et al.*[21] Case A. Ha.	49	15	− 11	+ 45	+ 46	+1.2
	Average		22.9				+3.6
	Androgen and Estrogen Together						
F	Reifenstein & Albright[98] Case 4	48	24	− 135	+ 173*	+ 308	+6.4
M	Reifenstein & Albright[98] Case 6a	70	20	+ 145	+ 424*	+ 279	+4.0
M	Reifenstein & Albright[98] Case 6b	69	30	+ 116	+ 191*	+ 75	+1.1
F	Shorr *et al*[111] Case E P.	70†	21	− 29	+ 629	+ 658	+9.4
	Average		24				+4.6

* First collection period (5-7 days) on therapy omitted.

† Data not reported; calculations based on 70 Kg. Reported weights averaged approximately 60 Kg

Steroids and Dosage testosterone propionate, 25 to 50 mg /day by injection; testosterone pellets, 375 mg (first 21 days after implantation), 17-methyltestosterone, 40 to 100 mg./day by mouth; estradiol benzoate, 0 55 to 1.66 mg./day by injection; and diethylstilbestrol, 6 to 7 mg./day (average) by mouth.

NITROGEN RETAINED DURING THE FIRST 30 DAYS OF THE ADMINISTRATION OF RETENTION IN MG. PER KG. PER DAY)

Phosphorus				Nitrogen			
Control (Mg / 24 Hrs)	Retention (Mg / 24 Hrs.)	Net Retention (Mg./ 24 Hrs)	Net Retention (Mg./ Kg./Day)	Control (Mg / 24 Hrs)	Retention (Mg / 24 Hrs.)	Net Retention (Mg / 24 Hrs)	Net Retention (Mg./ Kg./Day)
ANDROGEN ALONE							
− 20	+ 209*	+ 229	+4.8	+1437	+3570*	+2133	+44.4
+ 28	+ 281*	+ 253	+5.0	+ 570	+3510*	+2940	+57.6
+ 109	+ 335*	+ 226	+3.2	+1060	+3480*	+2420	+34.6
+ 166	+ 261	+ 95	+1.4	+ 610	+2910	+2300	+32.9
+ 139	+ 311	+ 172	+2.5	+3130	+4040	+ 910	+13.0
......	......	......	+3.3	......	......	......	+37.0
ESTROGEN ALONE							
+ 4	+ 127*	+ 123	+2.4	+ 123	+ 765*	+ 642	+12.6
− 8	+ 102*	+ 110	+2.0	+ 200	+ 910*	+ 710	+13.1
− 2	+ 64*	+ 66	+0.9	− 565	+ 658*	+1223	+16.5
− 184	− 41*	+ 143	+3.9	− 80	+ 306*	+ 386	+10.4
+ 109	+ 193*	+ 84	+1.3	− 540	− 55*	+ 485	+ 7.2
+ 319	+ 524	+ 205	+2.9	+2650	+3440	+ 790	+11.3
+ 146	+ 141	− 5	−0.06	+3300	+2700	− 600	− 6.9
+ 164	+ 207	+ 43	+0.73	+3625	+3200	− 425	− 7.2
− 9	+ 18	+ 27	+0.55	+2233	+1467	− 766	−15.6
......	......	......	+1.9	......	......	......	+ 7.9
ANDROGEN AND ESTROGEN TOGETHER							
− 15	+ 212*	+ 227	+4.7	+1460	+3200*	+1740	+36.3
+ 109	+ 359*	+ 250	+3.6	+1060	+3100*	+2040	+29.1
+ 75	+ 216*	+ 141	+2.0	− 10	+1781*	+1791	+26.0
+ 319	+ 878	+ 559	+8.0	+2650	+6280	+3630	+51.9
......	......	......	+4.3	......	......	......	+35.0

TABLE 7. ANABOLIC STEROID THERAPY IN SENILE OSTEOPOROSIS: COMPARISON OF CALCULATED (THEORETIC) RETENTION OF PHOSPHORUS ($P_{CALCIUM}$ + $P_{NITROGEN}$) WITH MEASURED RETENTION OF PHOSPHORUS DURING FIRST 30 DAYS OF ADMINISTRATION OF LARGE DOSES (SEE TABLE 6)

HORMONE THERAPY	A $P_{calcium}$ $\left[\frac{calcium}{2.3}\right]$ Mg./Kg /Day	B $P_{nitrogen}$ $\left[\frac{nitrogen}{15}\right]$ Mg /Kg./Day	C THEORETIC PHOSPHORUS RETENTION [A + B] Mg./Kg./Day	D MEASURED PHOSPHORUS RETENTION Mg./Kg /Day	E DIFFERENCE: THEORETIC OVER MEASURED [C − D] Mg./Kg./Day
Androgen alone ..	+0.91	+2.47	+3.38	+3.34	+0.04
Estrogen alone ...	+1.56	+0.52	+2.08	+1.94	+0.14
Both together	+2.00	+2.33	+4.33	+4.34	−0.01

TABLE 8. ANABOLIC STEROID THERAPY IN SENILE OSTEOPOROSIS: THE QUANTITY AND TIME RELATIONSHIPS OF CALCIUM RETENTION (SEE TABLE 6)

	ANDROGEN ALONE	ESTROGEN ALONE	BOTH TOGETHER
Gain per Kg. per day	+ 2.1 mg.	+ 3.6 mg.	+ 4.6 mg.
Gain per 60 Kg.* per day	+126 mg.	+216 mg.	+276 mg.
Gain per patient* per 30 days	+ 3.8 Gm	+ 6.5 Gm.	+ 8 3 Gm.
Gain per patient* per yr.	+ 45.4 Gm.	+ 77.8 Gm.	+ 99.4 Gm
For 345 Gm.† per patient*:			
Gaining at a constant rate	7.6 yrs.	4.4 yrs.	3.5 yrs.
Gaining at 10% less each yr. ...	14.3 yrs.	5.5 yrs.	4 1 yrs.

* Average weight (60 Kg.) of patients with senile osteoporosis (Table 6)
† Minimum calcium gain recognizable by roentgenogram (assumed; Table 2)

bination have qualitatively identical effects as anabolic agents.

A calculation of the quantitative differences between the anabolic steroids (Tables 6 & 8) shows that estrogen alone induced 1.7 times as much calcium retention as androgen alone, and that both steroids together resulted in 1.3 times as much retention as estrogen alone, and 2.2 times as much retention as androgen alone. Thus, a person who weighs 60 Kg. (the average weight of the patients with senile osteoporosis whose data were employed in the calculations) will retain per day 126 mg. of calcium with *androgen alone*, 216 mg. with estrogen alone, and 276 mg. with both steroids together during the initial response to steroid therapy. The quantities accumulated in 30

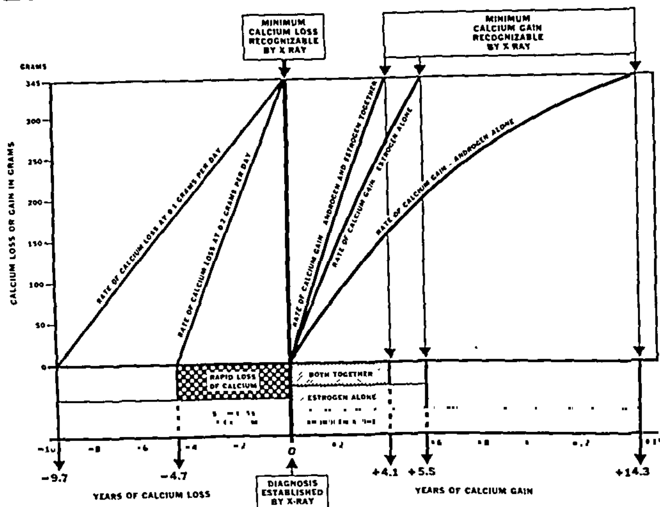


FIG. 15. The estimated time required in senile osteoporosis to produce roentgenographic evidence of the changes in the calcium content of bones that will permit (1) the recognition of the calcium loss and the establishment of the diagnosis and (2) the recognition of the calcium gain with anabolic steroid therapy.

The chart is derived from the data in Tables 2 and 8. The estimates are based upon (1) the observation that it requires a minimum calcium loss of 30 per cent (or 345 Gm.) before the decrease in the calcified bone mass can be recognized in roentgenograms[12,42,70,71] and (2) the assumptions that this same minimum amount of 30 per cent (i.e., 345 Gm.) must be restored to the skeleton before the increase can be recognized roentgenographically, and that the patient will gain 10 per cent less osseous tissues during each consecutive year of therapy.

It will be seen that on the basis of these estimates it will take approximately from 4.7 to 9.7 years for the calcium loss to be recognizable by roentgenogram. Furthermore, it will require 14.3 years with androgen alone, 5.5 years with estrogen alone and 4.1 years with both steroids together to add 345 Gm. of calcium to the skeleton. For further discussion see text.

days and in 1 year are sizable. In fact, the amount of calcium retained is so great that it cannot be accounted for unless it has been deposited in bone.

The time sequence of the calcium changes in senile osteoporosis is illustrated graphically in Figure 15 (based on Tables 2 & 8). We have estimated the time required to produce roentgenographic evidence that will permit (1) the recognition of the calcium loss and the establishment of the diagnosis and (2) the recognition of the calcium gain with anabolic steroid therapy. These estimates are based on the observation previously presented (see Table 2) that it requires a minimum calcium loss of 30 per

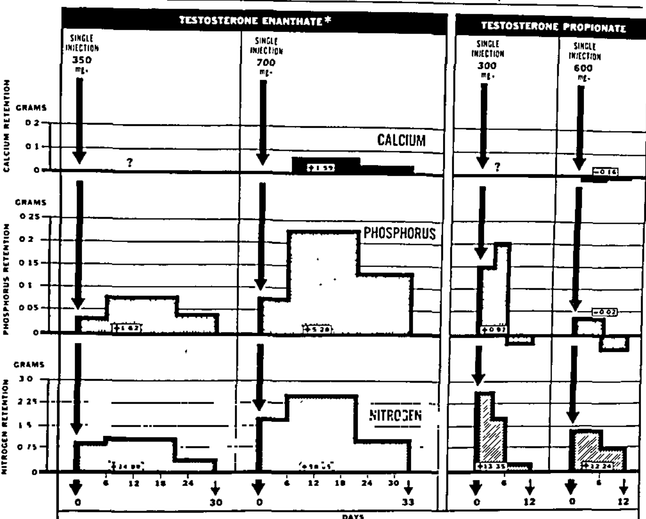


FIG. 16. Anabolic steroid therapy in osteoporosis (postmenopausal and senile): a comparison at two-dosage levels of the effect of one injection of testosterone enanthate with that of one injection of testosterone propionate upon the retention of calcium, phosphorus and nitrogen.

These metabolic balance studies of the effect of steroid compounds in osteoporosis were carried out on women aged 49 and 68. The scales for the balances in grams per day are given as the ordinates; the scale for time in days is given as the abscissa. The balances are charted as deviations from the average of the control periods rather than as the balances actually measured. Balances extending from the baseline toward the top of the chart are positive; those extending toward the bottom of the chart are negative. The scales are chosen so that 1 unit of phosphorus is equal to 15 units of nitrogen and 2 units of calcium to express the constant interrelationship of these substances that exist in normal protein and osseous tissues; thus the charted area of the phosphorus balance should equal the sum of the charted areas of the nitrogen and the calcium balances when protein and osseous tissues have been retained. The total retention in each balance during the interval in which the nitrogen balance is positive is given in the rectangles. The data are given in Table 9. At each of the two-dosage levels the testosterone enanthate and the testosterone propionate were given in amounts that contained equivalent quantities of testosterone.

It will be seen that testosterone enanthate at both dosage levels had a more marked and prolonged anabolic effect than testosterone propionate. The larger dose of the enanthate ester caused a much greater duration and degree of protein anabolism than an equivalent dosage of the propionate ester, and a slightly longer and considerably greater effect than the smaller amount of testosterone enanthate. Testosterone propionate had very little effect upon the retention of phosphorus and of calcium. In contrast, testosterone en[illegible] ...ant retention of phosphorus, and at the lar[illegible] ...lcium. For further discussion see text.

TABLE 9. ANABOLIC STEROID THERAPY IN SENILE OSTEOPOROSIS: A COMPARISON AT TWO-DOSAGE LEVELS OF THE EFFECT OF ONE INJECTION OF TESTOSTERONE PROPIONATE WITH THAT OF ONE INJECTION OF TESTOSTERONE ENANTHATE UPON THE RETENTION OF CALCIUM, PHOSPHORUS AND NITROGEN

Steroid Ester Given As Single Injection	Duration of Effect (Days)	Days of Collection	Calcium		Phosphorus		Nitrogen	
			Net Retention* (Mg./24 Hrs.)	Total Retention (Gm.)	Net Retention* (Mg./24 Hrs.)	Total Retention (Gm.)	Net Retention* (Gm./24 Hrs.)	Total Retention (Gm.)
TESTOSTERONE PROPIONATE								
Study No 1.... 300 mg.	12	1-3	—	—	+144	+ 0.43	+2.52	+ 7.56
		4-6	—	—	+196	+ 0.59	+1.75	+ 5.25
		7-12	—	—	− 9	− 0.05	+0.09	+ 0.54
Total............	....	....	....	—	....	+ 0.97	....	+13.35
Study No. 2.....600 mg.	12	1-6	− 21	− 0.13	+ 32	+ 0.19	+1.34	+ 8.04
		7-12	− 5	− 0.03	− 35	− 0.21	+0.70	+ 4.20
Total............	....	....	....	− 0.16	....	− 0.02	....	+12.24
TESTOSTERONE ENANTHATE†								
Study No. 1.....350 mg.	30	1-6	—	—	+ 29	+ 0.17	+0.89	+ 5.34
		7-21	—	—	+ 74	+ 1.11	+1.13	+16.95
		22-30	—	—	+ 38	+ 0.34	+0.30	+ 2.70
Total............	....	....	....	—	....	+ 1.62	....	+24.99
Study No. 2.....700 mg.	33	1-6	+ 16	+ 0.10	+ 72	+ 0.43	+1.70	+10.20
		7-21	+ 72	+ 1.08	+221	+ 3.31	+2.43	+36.45
		22-33	+ 34	+ 0.41	+128	+ 1.54	+0.95	+11.40
Total............	....	....	....	+ 1.59	....	+ 5.28	....	+58.05

* Data from Howard and Reifenstein[55]

† Delatestryl, E. R. Squibb and Sons, New York

cent (or 345 Gm.) before the decrease in the calcified bone mass can be recognized in roentgenograms.[12,42,70,71] If we assume that this same minimum amount of 30 per cent (that is, 345 Gm.) must be restored to the skeleton before the increase can be recognized roentgenographically, and that the patient will gain 10 per cent less osseous tissue during each consecutive year of therapy, it will require 14.3 years with androgen alone, 5.5 years with estrogen alone, and 4.1 years with both steroids together to add 345 Gm. of calcium to the skeleton. Therefore, it is not surprising that few reports have appeared in which indisputable evidence of increased calcified bone mass has been demonstrated in the roentgenograms following anabolic steroid therapy; few patients have been treated continuously with adequate dosage and for sufficient time to allow visualization of the calcium increment. If the diagnosis of senile osteoporosis is not established until a greater than 30 per cent calcium depletion has occurred, the time required to induce recognizable repletion with therapy may be prolonged considerably.

The previous discussion concerned anabolic steroids with relatively short action. We have made one preliminary study[55] of a steroid ester with a more prolonged duration of action, testosterone enanthate.*[59] We have compared at two-dosage levels the effect of one injection of testosterone propionate with that of one injection of testosterone enanthate upon the retention of calcium, phosphorus and nitrogen during metabolic balance studies in patients with senile osteoporosis (Fig. 16 & Table 9). At each of the two-dosage levels the esters were given in amounts that contained equivalent quantities of testosterone. The calcium retention was not measured for the smaller doses.

The single injection of 300 mg. of testosterone propionate had an effect which lasted 12 days, during which 13 Gm. of nitrogen was retained. Doubling the dose to 600 mg. as one injection did not increase the duration of the nitrogen retention beyond 12 days and also did not increase the total quantity (12 Gm.) which was retained. Phosphorus retention occurred with the smaller dose but not with the larger. The 600-mg. dose of the propionate ester had an insignificant effect upon calcium retention. In contrast, the administration of testosterone enanthate at both-dosage levels had a much more marked and prolonged anabolic effect. A single injection of 350 mg. of the enanthate ester had an action that persisted for 30 days, during which time 25 Gm. of nitrogen and over 1½ Gm. of phosphorus were retained. When the dose of testosterone enanthate was doubled to 700 mg. at one injection, the duration of anabolic action was increased to 33 days, and the total retention was more than doubled to 58 Gm. of nitrogen and over 5 Gm. of phosphorus. The most important effect was the significant retention of over 1½ Gm. of calcium. With the propionate ester, the greatest anabolic effect occurred during the first 3 to 6 days, while with the enanthate ester the maximal response did not appear until after 6 days. The potential of testosterone enanthate as a therapeutic agent for inducing bone anabolism in senile osteoporosis must be evaluated further. We have the impression that a still larger quantity as a single injection may have an even greater amount and duration of effect.

* Delatestryl, E. R. Squibb & Sons, New York

Secondary Evidence Supporting Question 1

Anabolic Steroid Deficiency in Presenile Persons. If a deficiency of anabolic steroids in aging persons leads to senile osteoporosis, a deficiency of these steroids in the presenile individual also should result in the occurrence of osteoporosis. This appears to be true. Osteoporosis develops about 20 years after the normal time of puberty in many patients with hypogonadism who have not received hormonal treatment.[8,15,69,80,81,84] Also, untreated patients with congenital absence of the gonads and the syndrome

TABLE 10. THE INCIDENCE OF OSTEOPOROSIS IN CUSHING'S SYNDROME

INVESTIGATORS	TOTAL NUMBER OF CASES	NUMBER OF CASES WITH OSTEOPOROSIS	PERCENTAGE OF CASES WITH OSTEOPOROSIS
Albright (reported by Irwin *et al.*[57])	43	39	91%
Eisenhardt and Thompson[36]	61	53	87%
Knowlton[65]	35	26	74%
Total	139	118	85%

of gonadal dysgenesis frequently have osteoporosis.[8,9,31,48,58,89,102,110,134] Furthermore, no cases of the osseous disorder have been encountered in patients with hypogonadism or gonadal dysgenesis who have received adequate gonadal steroid therapy.

Conclusions from Evidence on Question 1

The evidence concerning the first question leads to the following conclusions: (1) an absolute deficiency of anabolic steroid hormones occurs in aging persons; (2) this deficiency is one of the factors that leads to the development of senile osteoporosis in these aging individuals; (3) anabolic steroid therapy has a beneficial effect upon senile osteoporosis and tends to restore the calcified bone mass toward the normal state; and (4) anabolic steroid deficiency at any age is a factor that may lead to osteoporosis. Therefore, the first question may be answered by stating that there is an absolute deficiency of anabolic steroid hormones in senile osteoporosis.

QUESTION 2: IS THERE AN EXCESS OF ANTIANABOLIC STEROID HORMONES IN SENILE OSTEOPOROSIS?

The Nature of Antianabolic Steroids

The anterior pituitary adrenocorticotropic hormone (ACTH) and the adrenal cortical steroids (hydrocortisone [cortisol], cortisone and related compounds) have adverse effects upon the protein and the osseous tissues characterized by protein depletion and osteoporosis.[97] These hormones will be referred to hereafter as *corticoid steroids,* and also as *antianabolic steroids,* since we believe that in most circumstances corticoid hormones have an antianabolic rather than a catabolic action.[97]

Association of Osteoporosis with Excess of Antianabolic Steroids

Osteoporosis and Endogenous Excess of Antianabolic Steroids. Cushing's syndrome results from an excessive production and/or release of corticoid hormones.[1,7,61] Certain of the characteristic clinical features obviously involve depletion of the protein tissues; a negative nitrogen balance has been observed in some patients during balance studies.[1,7,61,121] Frequently, in addition, the excess corticoid hormones induce insufficient formation of protein matrix and, hence, inadequate formation of calcified bone. This results in osteoporosis, one of the most common serious manifestations of Cushing's syndrome. The incidence of bone involvement (Table 10) was 85 per cent in 139 cases.[36,57,65] The osteoporosis appears as soon as several months after the pathologic state is recognized. The manifestations include back pain; normal serum levels for calcium, inorganic phosphorus and alkaline phosphatase; hypercalciuria in most patients; generalized increased radiolucency of the bones most marked in the spine and the ribs; and collapsed vertebrae, most commonly in the lumbar and the thoracic areas. The osteoporosis of Cushing's syndrome is similar in all respects to that observed in senile osteoporosis.

Osteoporosis and Exogenous Excess of Antisteroids. The manifestations of Cush-

TABLE 11. THE AMOUNT AND THE DURATION OF CORTICOID THERAPY BEFORE SPONTANEOUS FRACTURE IN CHILDREN AND ADULTS OF BOTH SEXES

TYPE OF PATIENT	NUMBER OF PATIENTS	AGE AVERAGE AND RANGE (Yrs.)	TOTAL THERAPY* BEFORE FRACTURE AVERAGE AND RANGE (Gm)	DURATION OF THERAPY BEFORE FRACTURE AVERAGE AND RANGE (Days)
Girls	2	4.5 (3-6)	5 3 (4.9-5.72)	97 (76-119)
Boys	4	8.5 (3-11)	22.8 (7.09-47.5)	279 (71-478)
Women	9	56.2 (45-70)	30.5 (3 4-72.9)	352 (34-865)
	10	Not stated	Not stated	Not stated
Total	19	—	—	—
Men	8	61.1 (48-72)	42.7 (7.3-88.2)	605 (73-1148)
	1	Not stated	Not stated	Not stated
Total	9	—	—	—
Not stated	3	Not stated	17.3 (7.0-26.8)	162 (70-215)
	1	Not stated	3.5	Not stated
Total	4	—	—	—
Total cases	38	—	—	—

Adapted from the compilation of Reifenstein[97]

* Cortisone plus hydrocortisone plus adrenocorticotropic hormone (ACTH)

ing's syndrome arise from an endogenous excess of corticoid hormones. Therefore, it was anticipated that the same findings would be induced by an administered excess of corticoid homones if the therapy were given continuously in large amounts and/or for a sufficiently long period; almost all these manifestations have been observed.[17,22-24,38,39,57,62,85,101,117,118,121-123,128,130,131,140] As might be expected, the reported findings include adverse effects not only upon the protein tissues but also upon the osseous system with the development of osteoporosis, spontaneous fractures and hypercalciuria.

Recently, we surveyed[97] published reports[6,22 25,29,32,33,37,39,57,75,117,118,123,129,139,140] for evidence of disturbed bone metabolism following chronic corticoid medication. We found that after therapy for considerable periods of time and/or large doses, 38 cases had developed spontaneous fractures. The patients were receiving treatment for rheumatoid and related forms of arthritis, Still's disease, lupus erythematosus, pemphigus

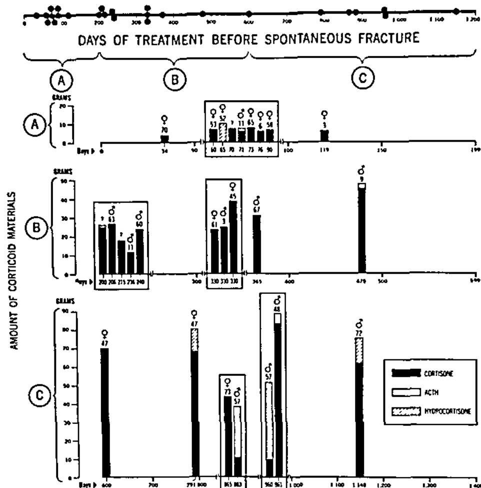


FIG. 17. The amount and the duration of corticoid therapy prior to spontaneous fracture in 26 patients with various disorders.

The scale for the total amount in grams of corticoid materials (cortisone, hydrocortisone and adrenocorticotropic hormone [ACTH]) administered prior to the occurrence of spontaneous fracture is given as the ordinate; the scale for the duration in days of corticoid therapy prior to the occurrence of spontaneous fracture is given as the abscissa. Note that there are four different time scales (top, A, B & C) and that the five areas enclosed in rectangles are enlarged horizontally but not vertically. Cortisone therapy is shown as a black bar; adrenocorticotropic hormone (ACTH) therapy as a white bar; and hydrocortisone therapy as a hatched bar. The sex and the age of the patient are given over each column of therapy.

The figure has 4 divisions: *(Top)* the distribution of spontaneous fractures in relation to duration of treatment on a continuous time scale: (A, *side*) the amount of medication and the occurrence of spontaneous fractures during therapy from 0 to 199 days; (B, *side*) the amount of medication and the occurrence of spontaneous fractures during corticoid therapy from 200 to 599 days; and (C, *side*) the amount of medication and the occurrence of spontaneous fractures from 600 to 1,400 days. It will be seen that the occurrence of spontaneous fractures is related to both the amount and the duration of therapy with corticoid materials, and that spontaneous fractures may result from treatment with cortisone, hydrocortisone or ACTH. These findings are evidence that in some patients corticoid therapy has an adverse effect on osseous tissue. For additional data on these patients see Table 11 For further discussion see text.

(Reifenstein, E. C., Jr., South. M. J. 49:933-960)

vulgaris, asthma and leukemia. Additional cases that did not fracture were reported to have developed osteoporosis. The amount and the duration of corticoid therapy prior to fracture in 26 of these patients are shown graphically in Figure 17. The data on the complete series are given in Table 11. The occurrence of spontaneous fractures was related both to the amount and to the duration of therapy, and resulted from treatment with all three corticoid medications alone or in combination. Before fracture the average dosage of cortisone was 26.1 Gm.; of hydrocortisone, 11.7 Gm.; and of ACTH, 11.6 Gm. The duration of therapy before fracture ranged from approximately 1 month to over 3 years. In the 28 adults, fractures occurred in over two times as many females as males; the vertebrae were involved much more frequently than other bones. We con-

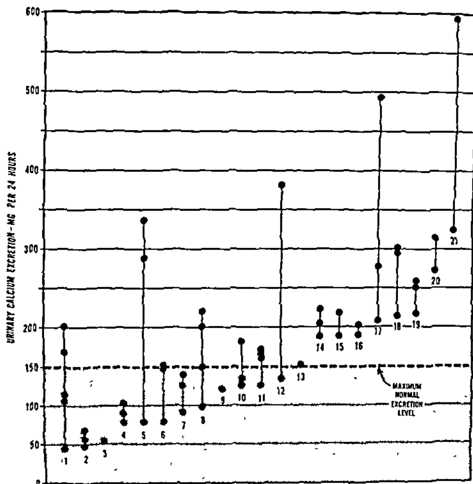


FIG. 18. The urinary calcium excretion of 21 patients with asthma on prolonged cortisone therapy.

The scale for the urinary calcium excretion in mg. per 24 hours is given as the ordinate; the individual patients are numbered consecutively in order of increasing calcium excretion along the abscissa. Each black dot represents a single determination of the urinary calcium excreted per 24 hours; all determinations for an individual patient are connected by a vertical line. The horizontal line of dashes at 150 mg. represents the level of excretion per 24 hours, which usually is maximal for normal subjects; the normal range of excretion is given as the stippled area Each patient required at least 600 mg. of cortisone to become cleared of asthma, and from 50 to 150 mg. of cortisone each day to be maintained in a symptom-free state. In most patients the cortisone was administered intramuscularly. The calcium determinations were made when the patients were on a maintenance regimen. The amount of cortisone therapy administered prior to each calcium determination and the order of and the interval between repeated determinations on the same patient are not stated by the authors.

It will be seen that in two thirds of the patients there was at least one elevated urinary calcium value and that in over one third of them there were urinary calcium excretion values above the normal maximal level. More than half of the determinations gave values in the hypercalciuric range. These findings are evidence that in some patients corticoid therapy has an adverse effect on osseous tissue.

(Data recharted from Figure 4 in Irwin *et al.*: J. Allergy 25:201-209)

cluded that spontaneous fractures following corticoid therapy were prone to occur particularly in (1) patients with pre-existing osteoporosis; (2) persons of middle and advanced age, (3) postmenopausal women; (4) children; (5) individuals with marked restriction of physical activity or enforced bed rest; and (6) patients who were given corticoid materials in large amounts and/or for prolonged periods.

The disturbed bone metabolism during chronic corticoid medication is indicated also by persistent hypercalciuria.[57] The urinary calcium excretion of 21 asthmatic patients on corticoid therapy is shown in Figure 18. Each patient required at least 600 mg of cortisone to become cleared of asthma and from 50 to 150 mg. of cortisone each day to be maintained in a symptom-free state. The calcium determinations were made when the patients were on a maintenance regimen. Two thirds of the patients had at least one

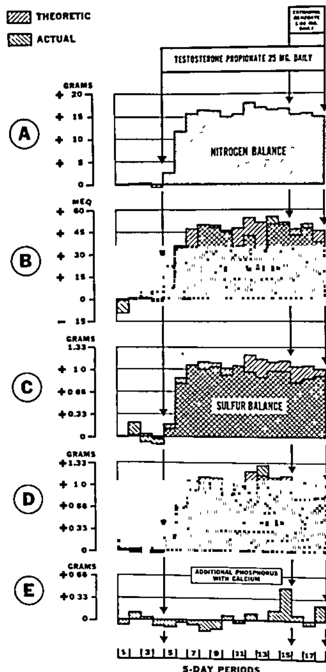


FIG. 19. The effect of anabolic steroid therapy upon the nitrogen and the mineral balances of a woman with Cushing's syndrome and osteoporosis.

This metabolic balance study of a 25-year-old woman with Cushing's syndrome and osteoporosis consists of 18 5-day periods. The scales for the balances in amounts per 5-day period are given as the ordinates; the scale of the 5-day periods is given as the abscissa. The horizontal line starting at zero on each ordinate is the baseline of that particular balance; balances extending from the baseline toward the tops of the diagrams are positive; those extending from the baseline toward the bottoms of the diagrams are negative. The balances are charted as deviations from the average of the control periods rather than as the balances actually measured. The scales are chosen so that 15 units of nitrogen are equal to 3 units (in grams; 45 units in milliequivalents) of potassium to 1 unit of phosphorus and of 1 unit of sulfur to express the constant interrelationships that exist in normal protein tissues; thus, the charted areas of the potassium balance, of the phosphorus balance (after the phosphorus theoretically retained with calcium has been subtracted) and of the sulfur balance in each instance should equal the charted area of the nitrogen balance when protein tissues have been retained. The data for potassium are based on analyses of urinary excretions alone, the fecal potassium excretion was assumed to be 8 per cent of the potassium intake The factors for the calculations are given in Table 8 in Albright and Reifenstein;[8] the methods for the accumulation, the presentation and the interpretation of these data are discussed in detail in Reifenstein, Albright and Wells.[99] Testosterone propionate and estradiol benzoate were administered intramuscularly each day during the periods indicated.

The figure has 5 divisions: (A) the measured nitrogen balance; (B) the measured nitrogen balance with superimposed theoretic nitrogen balance explainable by the measured potassium balance; (C) the measured nitrogen balance with superimposed theoretic nitrogen balance explainable by the measured phosphorus balance (after the phosphorus theoretically retained with calcium has been subtracted), (D) the measured nitrogen balance with superimposed theoretic nitrogen balance explainable by the measured sulfur balance; and (E) the theoretic balance of phosphorus explainable by the measured calcium balance

It will be seen that there is a close correspondence between the measured and the theoretic nitrogen balances This is evidence that testosterone propionate therapy induced a retention of nitrogen, potassium, phosphorus, sulfur and calcium in the proportions that exist in protein and osseous tissues Furthermore, the estradiol benzoate induced no significant alteration in retention of protein constituents when it was added to the testosterone propionate during periods 16, 17 and 18; estradiol benzoate was not given for a sufficient time to evaluate its effect upon the slowly responding calcium balance. For further discussion see text.

(Data from Reifenstein *et al.*: J. Clin. Endocrinol. 5:367-395. For further data on this patient see Case 37 in Fraser *et al.*[41] and Albright;[1-3] Case 1 in Albright *et al.*;[7] Case 10 in Reifenstein & Albright;[98] and Case 15 [Figs. 82 & 156] in Albright & Reifenstein[8])

elevated urinary calcium value, over one third had all their values above the normal maximal level and more than half of the values were in the hypercalciuric range. These findings are evidence that corticoid therapy tends to have an adverse effect upon osseous tissue.

Effect of Anabolic Steroid Therapy on Osteoporosis Associated with Excess of Antianabolic Steroids. The effect of anabolic steroid therapy in patients who have osteoporosis associated with excessive amounts of antianabolic steroids now will be considered. It had been postulated by Albright[7] that anabolic steroids would counteract the adverse effects of corticoid hormones upon the protein and the osseous tissues. A number of reports provide substantial evidence that this concept is correct.

The administration of androgenic compounds to patients with Cushing's syndrome has resulted in clinical improvement;[1-3,7,8,18,30,49,58,61,64,96,98,121,142] similar but less pronounced clinical benefit has been observed with estrogenic hormones.[8,34,35,44,83,88,93,96,98] The favorable effect of androgen has been demonstrated in patients with Cushing's syndrome who were fed amino acids containing radioactive nitrogen.[28,82] Before the steroid was administered, the radioactive nitrogen was excreted in the urine in about twice the normal quantity; after the hormone was given, the excretion fell to the normal level. This indicates that the anabolic steroid caused larger amounts of the ingested amino acids to be utilized for protein synthesis and, hence, reduced the amounts available for degradation and excretion. In patients with Cushing's syndrome, it has been demonstrated by balance studies that anabolic steroids cause growth of protein and osseous tissues.[1-3,7,8,30,66,82,88,121,122,128]

The effect of androgen alone and in combination with estrogen upon the nitrogen and the mineral balances of a 25-year-old woman with Cushing's syndrome and osteoporosis[1,7,8,98,99] is illustrated in Figure 19. The hormonal preparations induced a retention of over 3 Gm. of nitrogen per day and, simultaneously, a retention of sufficient quantities of potassium, sulfur, phosphorus and calcium to approximate their proportions in protein and osseous tissues for this amount of retained nitrogen. The calcium balance did not become positive until after 30 days of treatment; hence, estradiol benzoate was not given for a sufficient time to evaluate its effect upon the slowly responding calcium balance. There is a close correspondence between the measured and the theoretic balances. This is evidence that the anabolic steroid therapy induced anabolism of protein and osseous tissues.

Patients with a variety of clinical conditions are being maintained on continuous corticoid therapy for months or even years. We have pointed out previously that many of these individuals develop manifestations of protein depletion and osteoporosis.[17,22-24,38,39,57,62,85,101,117,118,121-123,128,130,131,140] Thus far, the simultaneous administration of anabolic steroids has been employed infrequently in such patients, and then only for relatively short periods.

The effect of corticoid therapy alone and in combination with androgen upon the urinary calcium excretion of a 60-year-old man with bronchial asthma and osteoporosis[24,57] is illustrated in Figure 20. Observations were made for more than 18 months. The urinary calcium excretion was considerably elevated in May, June and July, 1953, after the patient had been on continuous cortisone therapy for approximately 1 year. The calcium excretion fell to the normal range as soon as a sufficient amount (50 mg. per day) of testosterone propionate was administered along with the corticoid therapy; the excretion became elevated again as soon as the androgenic therapy was omitted; and the excretion returned to the normal range as soon as the corticoid therapy was reduced to low doses or omitted. This is evidence that the simultaneous administration of the anabolic steroid eliminated and/or prevented the adverse effect of chronic corticoid ther-

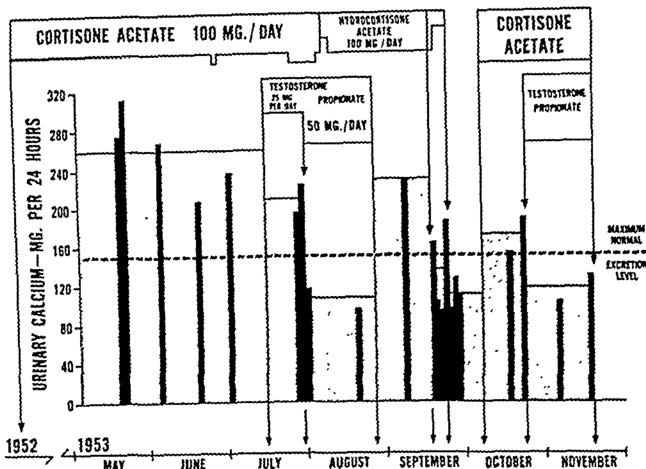


FIG. 20. The effect of anabolic steroid (testosterone propionate) therapy upon the hypercalciuria induced by chronic corticoid therapy in an elderly man with asthma and osteoporosis.

This study of a 60-year-old man with bronchial asthma and osteoporosis consisted of observations over more than 18 months. The scale for the urinary calcium excretion in mg. per 24 hours is given as the ordinate; the dates of treatment are given as the abscissa. The horizontal line of dashes at 150 mg. represents the level of excretion per 24 hours, which usually is maximal for normal subjects The average level of excretion during each period of study is shown by a horizontal line enclosing a stippled area. Cortisone acetate and hydrocortisone acetate were administered orally or intramuscularly in the dosage indicated; testosterone propionate was administered intramuscularly in the dosage indicated.

It will be seen that the urinary calcium excretion was considerably elevated in May, June and July, 1953, after the patient had been on continuous cortisone therapy for approximately 1 year. Note that the calcium excretion fell to the normal range as soon as a sufficient amount (50 mg per day) of testosterone propionate was administered along with the corticoid therapy; that the excretion became elevated again as soon as the androgenic hormone was omitted; and that the excretion returned to the normal range as soon as the corticoid therapy was reduced to low doses or omitted. This is evidence that the simultaneous administration of the anabolic steroid, testosterone propionate, eliminates and/or prevents the adverse effect of chronic corticoid therapy upon osseous tissue and calcium metabolism. Not indicated in the figure is the fact that the control of the asthmatic condition that had been achieved by the use of the corticoid substances was not interfered with by the simultaneous administration of the androgen For further discussion see text.

(Data recharted from Figure 3 in Irwin *et al.*: J. Allergy 25:201-209. For further data on this patient see Burrage *et al.*: Ann. Int. Med. 43:1001-1018)

elevated urinary calcium value, over one third had all their values above the normal maximal level and more than half of the values were in the hypercalciuric range. These findings are evidence that corticoid therapy tends to have an adverse effect upon osseous tissue.

Effect of Anabolic Steroid Therapy on Osteoporosis Associated with Excess of Antianabolic Steroids. The effect of anabolic steroid therapy in patients who have osteoporosis associated with excessive amounts of antianabolic steroids now will be considered. It had been postulated by Albright[7] that anabolic steroids would counteract the adverse effects of corticoid hormones upon the protein and the osseous tissues. A number of reports provide substantial evidence that this concept is correct.

The administration of androgenic compounds to patients with Cushing's syndrome has resulted in clinical improvement;[1-3,7,8,18,30,49,56,61,64,96,98,121,142] similar but less pronounced clinical benefit has been observed with estrogenic hormones.[8,34,35,44,83,88,93,96,98] The favorable effect of androgen has been demonstrated in patients with Cushing's syndrome who were fed amino acids containing radioactive nitrogen.[28,82] Before the steroid was administered, the radioactive nitrogen was excreted in the urine in about twice the normal quantity; after the hormone was given, the excretion fell to the normal level. This indicates that the anabolic steroid caused larger amounts of the ingested amino acids to be utilized for protein synthesis and, hence, reduced the amounts available for degradation and excretion. In patients with Cushing's syndrome, it has been demonstrated by balance studies that anabolic steroids cause growth of protein and osseous tissues.[1-3,7,8,30,66,82,88,121,122,128]

The effect of androgen alone and in combination with estrogen upon the nitrogen and the mineral balances of a 25-year-old woman with Cushing's syndrome and osteoporosis[1,7,8,98,99] is illustrated in Figure 19. The hormonal preparations induced a retention of over 3 Gm. of nitrogen per day and, simultaneously, a retention of sufficient quantities of potassium, sulfur, phosphorus and calcium to approximate their proportions in protein and osseous tissues for this amount of retained nitrogen. The calcium balance did not become positive until after 30 days of treatment; hence, estradiol benzoate was not given for a sufficient time to evaluate its effect upon the slowly responding calcium balance. There is a close correspondence between the measured and the theoretic balances. This is evidence that the anabolic steroid therapy induced anabolism of protein and osseous tissues.

Patients with a variety of clinical conditions are being maintained on continuous corticoid therapy for months or even years. We have pointed out previously that many of these individuals develop manifestations of protein depletion and osteoporosis.[17,22-24,38,39,57,62,85,101,117,118,121-123,128,130,131,140] Thus far, the simultaneous administration of anabolic steroids has been employed infrequently in such patients, and then only for relatively short periods.

The effect of corticoid therapy alone and in combination with androgen upon the urinary calcium excretion of a 60-year-old man with bronchial asthma and osteoporosis[24,57] is illustrated in Figure 20. Observations were made for more than 18 months. The urinary calcium excretion was considerably elevated in May, June and July, 1953, after the patient had been on continuous cortisone therapy for approximately 1 year. The calcium excretion fell to the normal range as soon as a sufficient amount (50 mg. per day) of testosterone propionate was administered along with the corticoid therapy; the excretion became elevated again as soon as the androgenic therapy was omitted; and the excretion returned to the normal range as soon as the corticoid therapy was reduced to low doses or omitted. This is evidence that the simultaneous administration of the anabolic steroid eliminated and/or prevented the adverse effect of chronic corticoid ther-

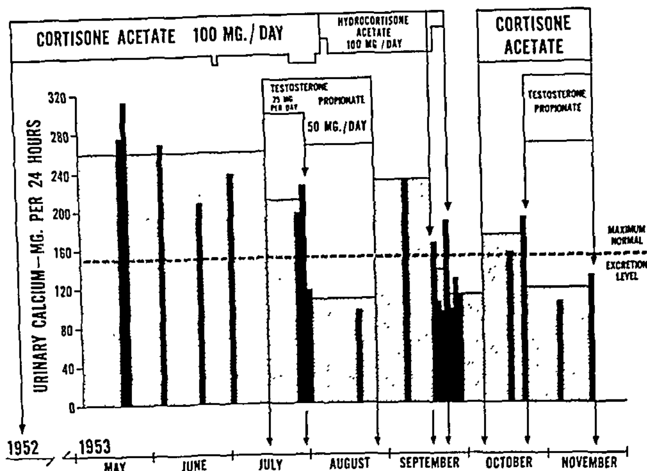


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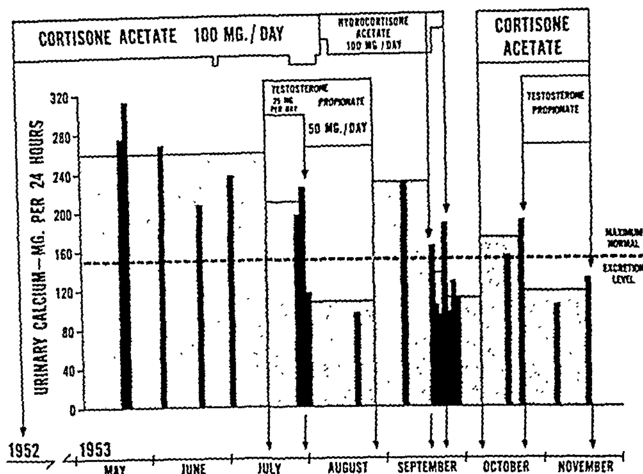


FIG. 20. The effect of anabolic steroid (testosterone propionate) therapy upon the hypercalciuria induced by chronic corticoid therapy in an elderly man with asthma and osteoporosis.

This study of a 60-year-old man with bronchial asthma and osteoporosis consisted of observations over more than 18 months. The scale for the urinary calcium excretion in mg. per 24 hours is given as the ordinate; the dates of treatment are given as the abscissa. The horizontal line of dashes at 150 mg represents the level of excretion per 24 hours, which usually is maximal for normal subjects. The average level of excretion during each period of study is shown by a horizontal line enclosing a stippled area. Cortisone acetate and hydrocortisone acetate were administered orally or intramuscularly in the dosage indicated; testosterone propionate was administered intramuscularly in the dosage indicated.

It will be seen that the urinary calcium excretion was considerably elevated in May, June and July, 1953, after the patient had been on continuous cortisone therapy for approximately 1 year. Note that the calcium excretion fell to the normal range as soon as a sufficient amount (50 mg. per day) of testosterone propionate was administered along with the corticoid therapy; that the excretion became elevated again as soon as the androgenic hormone was omitted, and that the excretion returned to the normal range as soon as the corticoid therapy was reduced to low doses or omitted. This is evidence that the simultaneous administration of the anabolic steroid, testosterone propionate, eliminates and/or prevents the adverse effect of chronic corticoid therapy upon osseous tissue and calcium metabolism. Not indicated in the figure is the fact that the control of the asthmatic condition that had been achieved by the use of the corticoid substances was not interfered with by the simultaneous administration of the androgen. For further discussion see text.

(Data recharted from Figure 3 in Irwin *et al.*: J. Allergy 25:201-209. For further data on this patient see Burrage *et al.*: Ann. Int. Med. 43:1001-1018)

elevated urinary calcium value, over one third had all their values above the normal maximal level and more than half of the values were in the hypercalciuric range. These findings are evidence that corticoid therapy tends to have an adverse effect upon osseous tissue.

Effect of Anabolic Steroid Therapy on Osteoporosis Associated with Excess of Antianabolic Steroids. The effect of anabolic steroid therapy in patients who have osteoporosis associated with excessive amounts of antianabolic steroids now will be considered. It had been postulated by Albright[7] that anabolic steroids would counteract the adverse effects of corticoid hormones upon the protein and the osseous tissues. A number of reports provide substantial evidence that this concept is correct.

The administration of androgenic compounds to patients with Cushing's syndrome has resulted in clinical improvement;[1-3,7,8,18,30,40,56,61,64,96,98,121,142] similar but less pronounced clinical benefit has been observed with estrogenic hormones.[8,34,35,44,83,88,93,96,99] The favorable effect of androgen has been demonstrated in patients with Cushing's syndrome who were fed amino acids containing radioactive nitrogen.[29,82] Before the steroid was administered, the radioactive nitrogen was excreted in the urine in about twice the normal quantity; after the hormone was given, the excretion fell to the normal level. This indicates that the anabolic steroid caused larger amounts of the ingested amino acids to be utilized for protein synthesis and, hence, reduced the amounts available for degradation and excretion. In patients with Cushing's syndrome, it has been demonstrated by balance studies that anabolic steroids cause growth of protein and osseous tissues.[1-3,7,8,30,66,82,88,121,122,128]

The effect of androgen alone and in combination with estrogen upon the nitrogen and the mineral balances of a 25-year-old woman with Cushing's syndrome and osteoporosis[1,7,8,98,99] is illustrated in Figure 19. The hormonal preparations induced a retention of over 3 Gm. of nitrogen per day and, simultaneously, a retention of sufficient quantities of potassium, sulfur, phosphorus and calcium to approximate their proportions in protein and osseous tissues for this amount of retained nitrogen. The calcium balance did not become positive until after 30 days of treatment; hence, estradiol benzoate was not given for a sufficient time to evaluate its effect upon the slowly responding calcium balance. There is a close correspondence between the measured and the theoretic balances. This is evidence that the anabolic steroid therapy induced anabolism of protein and osseous tissues.

Patients with a variety of clinical conditions are being maintained on continuous corticoid therapy for months or even years. We have pointed out previously that many of these individuals develop manifestations of protein depletion and osteoporosis.[17,22-24,38,39,57,62,85,101,117,118,121-123,128,130,131,140] Thus far, the simultaneous administration of anabolic steroids has been employed infrequently in such patients, and then only for relatively short periods.

The effect of corticoid therapy alone and in combination with androgen upon the urinary calcium excretion of a 60-year-old man with bronchial asthma and osteoporosis[24,57] is illustrated in Figure 20. Observations were made for more than 18 months. The urinary calcium excretion was considerably elevated in May, June and July, 1953, after the patient had been on continuous cortisone therapy for approximately 1 year. The calcium excretion fell to the normal range as soon as a sufficient amount (50 mg. per day) of testosterone propionate was administered along with the corticoid therapy; the excretion became elevated again as soon as the androgenic therapy was omitted; and the excretion returned to the normal range as soon as the corticoid therapy was reduced to low doses or omitted. This is evidence that the simultaneous administration of the anabolic steroid eliminated and/or prevented the adverse effect of chronic corticoid ther-

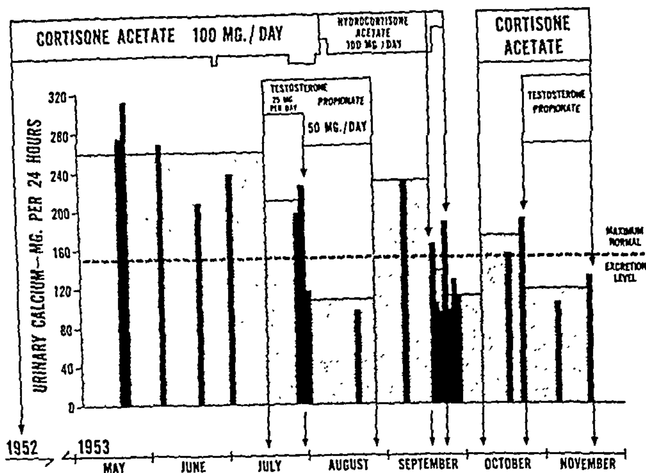


FIG. 20. The effect of anabolic steroid (testosterone propionate) therapy upon the hypercalciuria induced by chronic corticoid therapy in an elderly man with asthma and osteoporosis.

This study of a 60-year-old man with bronchial asthma and osteoporosis consisted of observations over more than 18 months. The scale for the urinary calcium excretion in mg. per 24 hours is given as the ordinate; the dates of treatment are given as the abscissa. The horizontal line of dashes at 150 mg. represents the level of excretion per 24 hours, which usually is maximal for normal subjects. The average level of excretion during each period of study is shown by a horizontal line enclosing a stippled area. Cortisone acetate and hydrocortisone acetate were administered orally or intramuscularly in the dosage indicated; testosterone propionate was administered intramuscularly in the dosage indicated

It will be seen that the urinary calcium excretion was considerably elevated in May, June and July, 1953, after the patient had been on continuous cortisone therapy for approximately 1 year. Note that the calcium excretion fell to the normal range as soon as a sufficient amount (50 mg per day) of testosterone propionate was administered along with the corticoid therapy, that the excretion became elevated again [illegible] androgenic [illegible] soon as the [illegible] the simulta[illegible] of the anabolic steroid, testosterone propionate, eliminates and/or prevents the adverse effect of chronic corticoid therapy upon osseous tissue and calcium metabolism Not indicated in the figure is the fact that the control of the asthmatic condition that had been achieved by the use of the corticoid substances was not interfered with by the simultaneous administration of the androgen. For further discussion see text.

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Patients with a variety of clinical conditions are being maintained on continuous corticoid therapy for months or even years. We have pointed out previously that many of these individuals develop manifestations of protein depletion and osteoporosis.[17,22-24,38,39,57,62,85,101,117,118,121-123,128,130,131,140] Thus far, the simultaneous administration of anabolic steroids has been employed infrequently in such patients, and then only for relatively short periods.

The effect of corticoid therapy alone and in combination with androgen upon the urinary calcium excretion of a 60-year-old man with bronchial asthma and osteoporosis[24,57] is illustrated in Figure 20. Observations were made for more than 18 months. The urinary calcium excretion was considerably elevated in May, June and July, 1953, after the patient had been on continuous cortisone therapy for approximately 1 year. The calcium excretion fell to the normal range as soon as a sufficient amount (50 mg. per day) of testosterone propionate was administered along with the corticoid therapy; the excretion became elevated again as soon as the androgenic therapy was omitted; and the excretion returned to the normal range as soon as the corticoid therapy was reduced to low doses or omitted. This is evidence that the simultaneous administration of the anabolic steroid eliminated and/or prevented the adverse effect of chronic corticoid ther-

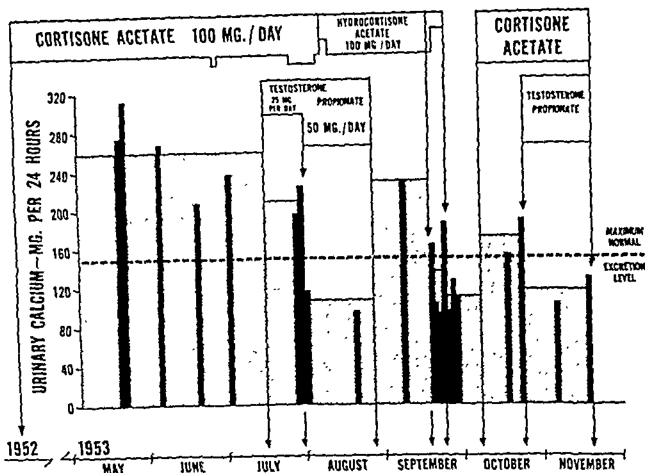


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The effect of androgen alone and in combination with estrogen upon the nitrogen and the mineral balances of a 25-year-old woman with Cushing's syndrome and osteoporosis[1,7,8,98,99] is illustrated in Figure 19. The hormonal preparations induced a retention of over 3 Gm. of nitrogen per day and, simultaneously, a retention of sufficient quantities of potassium, sulfur, phosphorus and calcium to approximate their proportions in protein and osseous tissues for this amount of retained nitrogen. The calcium balance did not become positive until after 30 days of treatment; hence, estradiol benzoate was not given for a sufficient time to evaluate its effect upon the slowly responding calcium balance. There is a close correspondence between the measured and the theoretic balances. This is evidence that the anabolic steroid therapy induced anabolism of protein and osseous tissues.

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The effect of corticoid therapy alone and in combination with androgen upon the urinary calcium excretion of a 60-year-old man with bronchial asthma and osteoporosis[24,57] is illustrated in Figure 20. Observations were made for more than 18 months. The urinary calcium excretion was considerably elevated in May, June and July, 1953, after the patient had been on continuous cortisone therapy for approximately 1 year. The calcium excretion fell to the normal range as soon as a sufficient amount (50 mg. per day) of testosterone propionate was administered along with the corticoid therapy; the excretion became elevated again as soon as the androgenic therapy was omitted; and the excretion returned to the normal range as soon as the corticoid therapy was reduced to low doses or omitted. This is evidence that the simultaneous administration of the anabolic steroid eliminated and/or prevented the adverse effect of chronic corticoid ther-

TABLE 12. THE EFFECT OF AGE AND SEX ON URINARY EXCRETION OF STEROIDS WITH ANTIANABOLIC ACTIVITY (NEUTRAL REDUCING LIPIDS)

Age Range (Yrs.)	Neutral Reducing Lipids Sleeping* (Mg./Hrs.)	Waking* (Mg./Hrs.)	Day* (Mg./Hrs.)	Average (Mg./Hrs.)	A Neutral Reducing Lipids (% of Excretion of Men Aged 20-29)	B Adjusted Neutral Reducing Lipids (% of Column A Equivalent to % of Androgen Excretion of Men Aged 20-29) (from Table 3 Column E)
Women						
20-29	1.61	2.22	1.88	1.90	88%	57%
30-39	1.60	1.86	2.07	1.84	85%	55%
40-49	1.34	1.77	2.04	1.72	80%	52%
50-59	1.67	1.81	1.72	1.73	80%	52%
60-90	1.50	1.55	1.69	1.58	73%	48%
Men						
20-29	1.76	2.52	2.19	2.16	100%	65%
30-39	1.79	2.66	2.02	2.16	100%	65%
40-49	2.27	2.69	2.57	2.50	116%	75%
50-59	1.87	2.20	2.23	2.10	97%	63%
60-90	1.90	2.12	1.98	2.00	93%	60%

Age Range (Yrs.)	C Total Androgen plus Estrogen (from Table 3 Column D)	D Total Neutral Reducing Lipids Combined with Total Androgen plus Estrogen (Column B plus Column C)	E Neutral Reducing Lipids (% of Total in Column D)	F Total Androgen plus Estrogen (% of Total in Column D)	G Neutral Reducing Lipids Difference from Equilibrium with Anabolic Steroids (Difference of E from 50%)
Women					
20-29	100%	157%	36%	64%	−14%
30-39	90%	145%	38%	62%	−12%
40-49	44%	96%	54%	46%	+ 4%
50-59	28%	80%	65%	35%	+15%
60-90	19%	67%	72%	28%	+22%
Men					
20-29	87%	152%	43%	57%	− 7%
30-39	73%	138%	47%	53%	− 3%
40-49	49%	124%	60%	40%	+10%
50-59	46%	109%	58%	42%	+ 8%
60-90	42%	102%	59%	41%	+ 9%

* Data of Pincus, Dorfman and associates[90,92]

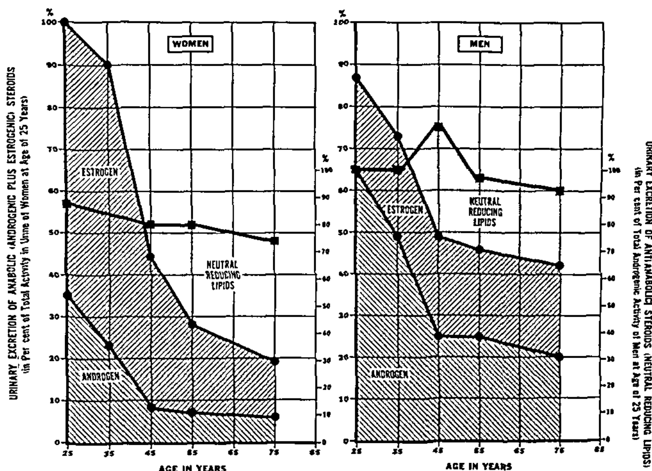


FIG. 21. The effect of age and sex upon the urinary excretion of steroids with anabolic activity (androgen and estrogen) and with antianabolic activity (neutral reducing lipids). The charts are derived from the data of Pincus, Dorfman and associates.[90-92] The graphs are the same as those given before in Figure 6 (see Table 3), but, in addition, data have been added for the antianabolic steroid excretion (see Table 12). The data were treated as follows: (1) the urinary excretion of neutral reducing lipids was used as an index of the antianabolic steroid excretion; (2) the approximate excretion at various ages was calculated for each sex by averaging the values for the sleeping, waking and day collections of the original study; (3) the urinary antianabolic activity excreted at various ages was calculated for each sex in percentage of the amount excreted by men at the age of 25 years; (4) then for each sex the activity values were recalculated as the percentage equivalent of the androgen excretion of men at the age of 25 years; and (5) the resulting values were charted. The urinary excretion of neutral reducing lipids is assumed to be an index, if not a measure, of the production of antianabolic steroids. The graphs show that with declining years (1) the production of antianabolic steroids diminishes only slightly in both sexes and (2) the more marked decrease in the production of anabolic steroids results in a relative excess of antianabolic steroids over anabolic steroids which is more rapidly induced and considerably greater in women than in men. Thus, the aging individual develops a steroid imbalance qualitatively similar to that of Cushing's syndrome For further discussion see text.

TABLE 12. THE EFFECT OF AGE AND SEX ON URINARY EXCRETION OF STEROIDS WITH ANTIANABOLIC ACTIVITY (NEUTRAL REDUCING LIPIDS)

Age Range (Yrs.)	Neutral Reducing Lipids: Sleeping* (Mg./Hrs.)	Waking* (Mg./Hrs.)	Day* (Mg./Hrs.)	Average (Mg./Hrs.)	A: Neutral Reducing Lipids (% of Excretion of Men Aged 20-29)	B: Adjusted Neutral Reducing Lipids (% of Column A Equivalent to % of Androgen Excretion of Men Aged 20-29) (from Table 3 Column E)
Women						
20-29	1.61	2.22	1.88	1.90	88%	57%
30-39	1.60	1.86	2.07	1.84	85%	55%
40-49	1.34	1.77	2.04	1.72	80%	52%
50-59	1.67	1.81	1.72	1.73	80%	52%
60-90	1.50	1.55	1.69	1.58	73%	48%
Men						
20-29	1.76	2.52	2.19	2.16	100%	65%
30-39	1.79	2.66	2.02	2.16	100%	65%
40-49	2.27	2.69	2.57	2.50	116%	75%
50-59	1.87	2.20	2.23	2.10	97%	63%
60-90	1.90	2.12	1.98	2.00	93%	60%

Age Range (Yrs.)	C: Total Androgen plus Estrogen (from Table 3 Column D)	D: Total Neutral Reducing Lipids Combined with Total Androgen plus Estrogen (Column B plus Column C)	E: Neutral Reducing Lipids (% of Total in Column D)	F: Total Androgen plus Estrogen (% of Total in Column D)	G: Neutral Reducing Lipids Difference from Equilibrium with Anabolic Steroids (Difference of E from 50%)
Women					
20-29	100%	157%	36%	64%	−14%
30-39	90%	145%	38%	62%	−12%
40-49	44%	96%	54%	46%	+ 4%
50-59	28%	80%	65%	35%	+15%
60-90	19%	67%	72%	28%	+22%
Men					
20-29	87%	152%	43%	57%	− 7%
30-39	73%	138%	47%	53%	− 3%
40-49	49%	124%	60%	40%	+10%
50-59	46%	109%	58%	42%	+ 8%
60-90	42%	102%	59%	41%	+ 9%

* Data of Pincus, Dorfman and associates[90, 92]

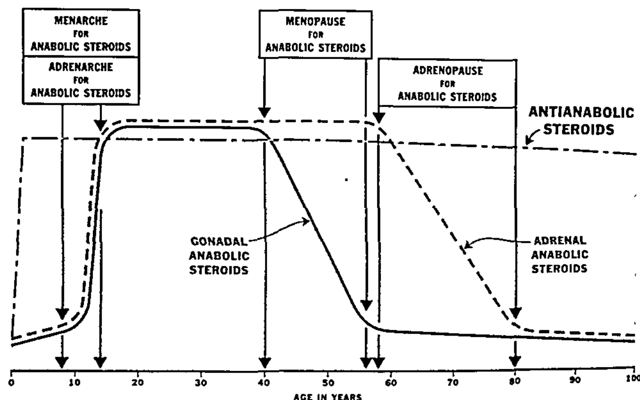


FIG. 22. The schematic life history of anabolic and antianabolic steroids in the human female.

This chart is the same as that in Figure 7 with additions. The life history of the anabolic steroids in the female is described in the caption for Figure 7. Steroids of the antianabolic group are first produced in a significant quantity very early in childhood, years before the first significant production of anabolic steroids. The production of antianabolic steroids decreases only slightly with age. For further discussion see text.

apy upon osseous tissue and calcium metabolism. It should be mentioned that the control of the asthmatic condition, which had been achieved by the use of the corticoid substances, was not interfered with by the simultaneous administration of the androgen.

Primary Evidence Supporting Question 2

Antianabolic Steroid Excess in Aging Persons. Certain of the clinical features of aging are similar to those observed in persons with an excess of corticoid hormones. As examples we can mention the thin skin, the reduced muscle mass, the decreased muscular power and weakness, and the ... tibility of the bones to fractur... the urinary excretion of antianab... metabolites is not elevated in ... it is in patients with Cushing's sy... in persons who are being treated ... coid medications.[126,127,135] Thus, th...

absolute excess of antianabolic hormones in senile osteoporosis.

Recent investigations[3,20,90-92,103,127] have established a very important point: with aging, the anabolic steroid production declines markedly, while in contrast the antianabolic steroid production decreases only very slightly. The general pattern of these changes in women and in men at various ages is shown graphically in Figure 21, which has been prepared from data (Table 12) of Pincus, Dorfman and associates.[90-92] The urinary excretion of neutral reducing lipids is assumed ... be an index, if not a measure, of th... of antianabolic steroids. In w... ...evel of the neutral reducing ... below that of men and ... but moderately ...oughout ... of the pro... ...ed fall ... steroi... after the ... is ...

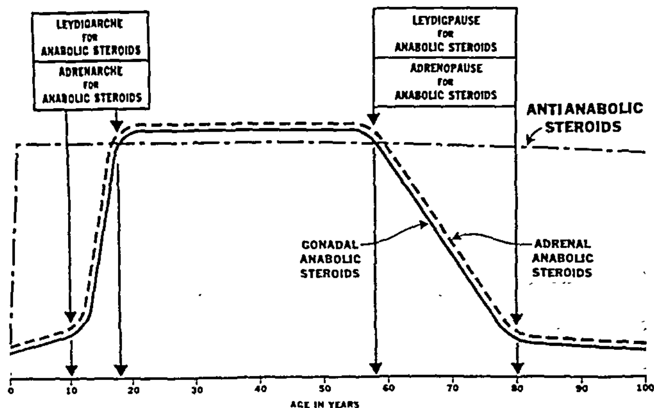


FIG. 23. The schematic life history of anabolic and antianabolic steroids in the human male.

This chart is the same as that given in Figure 8 with additions. The life history of the anabolic steroids in the male is described in the caption for Figure 8. Steroids of the antianabolic group are first produced in a significant quantity very early in childhood, years before the first significant production of anabolic steroids. The production of antianabolic steroids decreases only slightly with age. For further discussion see text.

relative excess of antianabolic steroids over anabolic steroids in the women during the latter years of life. In men, the initial level of neutral reducing lipids is a little higher than that of women, increases still further to a peak at about the age of 45, then declines even more gradually than that of women to a value at the end of life which is only slightly below the initial level. Because there is also a fall in the anabolic steroid excretion in men, there is likewise a relative excess of antianabolic steroids over anabolic steroids in the elderly male; however, the excess is not nearly as great as in women. Thus, the aging individual develops a steroid imbalance qualitatively similar to that of Cushing's syndrome.

The life history of the antianabolic steroid hormones in the female[3] is presented schematically in Figure 22, a chart shown previously as Figure 7 with certain additions. In the female, steroids of the antianabolic group are first produced in a significant quantity very early in childhood, years before the menarche and the adrenarche of the anabolic steroids. The production of antianabolic steroids decreases only slightly with age, so that a considerable amount still is being produced at the end of life. Therefore, there is no period of marked decline with age that corresponds to the menopause or the adrenopause with respect to the anabolic steroids.

The life history of the antianabolic steroid hormones in the male[3] is presented schematically in Figure 23, which is similar to Figure 8 with certain additions. In the male, the pattern of antianabolic steroid production is very similar to that in the female. A high level is reached in early childhood, and the amount produced declines only *slightly* with age, so that the level still is high at the

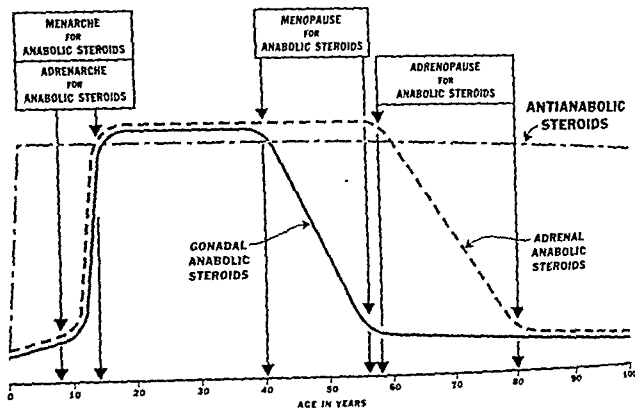


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apy upon osseous tissue and calcium metabolism. It should be mentioned that the control of the asthmatic condition, which had been achieved by the use of the corticoid substances, was not interfered with by the simultaneous administration of the androgen.

Primary Evidence Supporting Question 2

Antianabolic Steroid Excess in Aging Persons. Certain of the clinical features of aging are similar to those observed in persons with an excess of corticoid hormones. As examples we can mention the thin skin, the reduced muscle mass, the decreased muscular power and weakness, and the susceptibility of the bones to fracture. However, the urinary excretion of antianabolic steroid metabolites is not elevated in old people as it is in patients with Cushing's syndrome and in persons who are being treated with corticoid medications.[126,127,135] Thus, there is no absolute excess of antianabolic hormones in senile osteoporosis.

Recent investigations[3,20,90-92,103,127] have established a very important point: with aging, the anabolic steroid production declines markedly, while in contrast the antianabolic steroid production decreases only very slightly. The general pattern of these changes in women and in men at various ages is shown graphically in Figure 21, which has been prepared from data (Table 12) of Pincus, Dorfman and associates.[90-92] The urinary excretion of neutral reducing lipids is assumed to be an index, if not a measure, of the production of antianabolic steroids. In women, the level of the neutral reducing lipids is somewhat below that of men and declines steadily but moderately throughout life. As a result of the pronounced fall in the anabolic steroid excretion after the age of 35, there is a marked

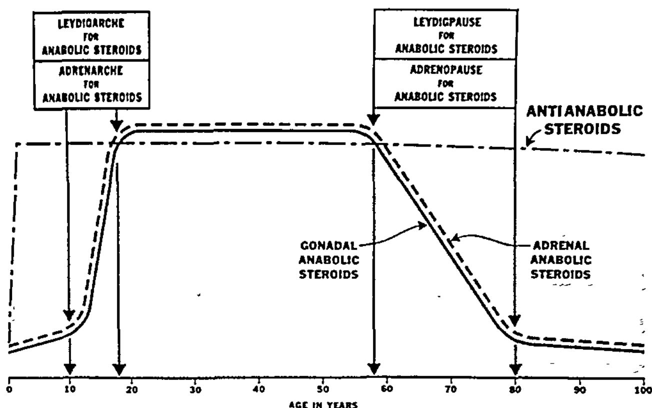


FIG. 23. The schematic life history of anabolic and antianabolic steroids in the human male.

This chart is the same as that given in Figure 8 with additions. The life history of the anabolic steroids in the male is described in the caption for Figure 8. Steroids of the antianabolic group are first produced in a significant quantity very early in childhood, years before the first significant production of anabolic steroids. The production of antianabolic steroids decreases only slightly with age. For further discussion see text.

relative excess of antianabolic steroids over anabolic steroids in the women during the latter years of life. In men, the initial level of neutral reducing lipids is a little higher than that of women, increases still further to a peak at about the age of 45, then declines even more gradually than that of women to a value at the end of life which is only slightly below the initial level. Because there is also a fall in the anabolic steroid excretion in men, there is likewise a relative excess of antianabolic steroids over anabolic steroids in the elderly male; however, the excess is not nearly as great as in women. Thus, the aging individual develops a steroid imbalance qualitatively similar to that of Cushing's syndrome.

The life history of the antianabolic steroid hormones in the female[3] is presented schematically in Figure 22, a chart shown previously as Figure 7 with certain additions. In the female, steroids of the antianabolic group are first produced in a significant quantity very early in childhood, years before the menarche and the adrenarche of the anabolic steroids. The production of antianabolic steroids decreases only slightly with age, so that a considerable amount still is being produced at the end of life. Therefore, there is no period of marked decline with age that corresponds to the menopause or the adrenopause with respect to the anabolic steroids.

The life history of the antianabolic steroid hormones in the male[3] is presented schematically in Figure 23, which is similar to Figure 8 with certain additions. In the male, the pattern of antianabolic steroid production is very similar to that in the female. A high level is reached in early childhood, and the amount produced declines only slightly with age, so that the level still is high at the

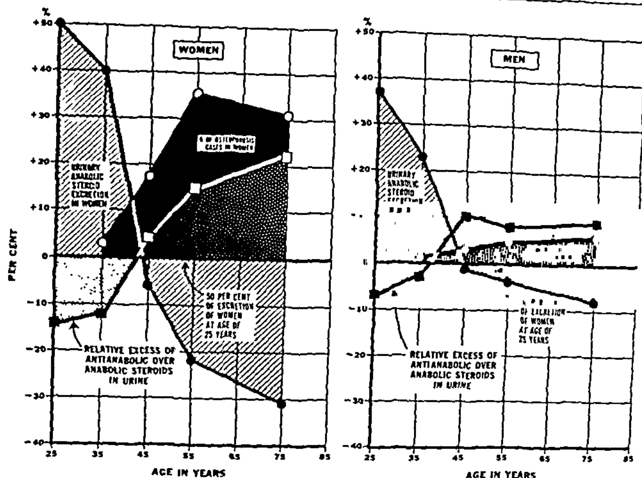


FIG. 24. The association in aging persons of senile osteoporosis with absolute anabolic steroid deficiency and with relative antianabolic steroid excess.

This chart is derived from the data in Figures 11 and 21.[10,26,87,90-92] The calculations are given in the caption for Figure 21; the data, in Tables 3 and 12. Data have been added for the difference between the neutral reducing lipid excretion and the total anabolic steroid excretion This difference was derived (1) by adding the percentage excretion of the neutral reducing lipids to that of the anabolic steroids for each of the respective age periods of each sex and then (2) by calculating the percentages of the components in the summation.

It will be seen that in women the rapid and marked increase in the cases of senile osteoporosis correlates in time not only with the abrupt fall in the absolute amount of anabolic steroids but also equally well with the abrupt rise in the relative amount of antianabolic steroids. In contrast, in men the more gradual and less pronounced increase in the cases of senile osteoporosis correlates in time not only with the more gradual and less marked fall in the absolute amount of anabolic steroids but equally well with the more gradual and less marked rise in the relative amount of antianabolic steroids For further discussion see text.

end of life. Therefore, there are no periods of changing production that correspond either to the leydigarche and the adrenarche or to the leydigpause and the adrenopause with respect to the anabolic steroids.

The time sequence of the excretion of anabolic and antianabolic steroids and of the occurrence of senile osteoporosis is presented graphically in Figure 24 This figure illustrates the point that in women the rapid and marked increase in the cases of senile osteoporosis correlates in time not only with the abrupt fall in the absolute amount of anabolic steroids but also equally well with the abrupt rise in the relative amount of antianabolic steroids. In contrast, in men

the more gradual and less pronounced increase in the cases of senile osteoporosis correlates in time not only with the more gradual and less marked fall in the absolute amount of anabolic steroids but also equally well with the more gradual and less marked rise in the relative amount of antianabolic steroids. Thus, osteoporosis is associated with the alterations in both types of steroid.

Conclusions from Evidence on Question 2

The evidence concerning the second question leads to the following conclusions: (1) a relative excess of antianabolic steroid hormones occurs in aging persons; (2) the imbalance between the endogenous antianabolic and anabolic steroids which results is qualitatively similar to that in Cushing's syndrome and in chronic corticoid therapy; (3) the absolute excess of antianabolic steroids in Cushing's syndrome and in chronic corticoid treatment results in the development of osteoporosis identical with senile osteoporosis in its manifestations and in its response to anabolic steroid therapy; (4) the time sequence of the development of senile osteoporosis correlates with the occurrence of the relative excess of antianabolic hormones in aging persons; and (5) this relative excess is one of the factors that leads to the development of senile osteoporosis in aging individuals. Therefore, the second question may be answered by stating that there is a relative excess of antianabolic steroid hormones in senile osteoporosis.

Recapitulation of Answers: There Is an Absolute Deficiency of Anabolic Steroid Hormones and a Relative Excess of Antianabolic Steroid Hormones in Senile Osteoporosis

The major relationships of steroid hormones to senile osteoporosis have been discussed by considering two questions, it is time to restate and integrate the answers. The evidence reviewed indicates that there is an absolute deficiency of anabolic steroid hormones *and at the same time* a relative excess of antianabolic steroid hormones in senile osteoporosis. The latter alteration appears to be the more important for the development of the bone condition. This is suggested by the observation that an absolute deficiency of anabolic steroids without a relative excess of antianabolic hormones apparently does not lead to osteoporosis. For example, we are not aware of any reports of this bone disorder in patients with panhypopituitarism,[3] in whom there is an absolute deficiency of the anabolic steroids and also, at the same time, of the antianabolic hormones. The relative excess of antianabolic steroids probably is an important factor also in the protein depletion of aging persons. This interpretation receives support from the recent studies of Sobel and Marmorston.[113-116] These investigators showed that in rats the ratio of the mucopolysaccharide constituent, hexosamine, to the collagen in the skin and the bones is decreased with age, is decreased in young adult rats by the administration of cortisone acetate and is maintained at the ratio of the young adult rat in aging animals by the administration of an anabolic steroid such as testosterone

DISCUSSION

The concept of the balance between anabolic and antianabolic steroids in various physiologic and pathologic states is illustrated schematically in Figure 25. The chart shows a balance between the hormonal influences in the normal adult (A); an absolute increase in the antianabolic activity in Cushing's syndrome and in the previously normal adult receiving chronic corticoid therapy (B & C); and a relative increase in the antianabolic activity (as a result of a decrease in the anabolic activity) in old age, in postmenopausal women and in hypogonadism with normal adrenal cortical function (D, E & F). The normal child has a level of anabolic steroids that is low for a mature individual but has an adult amount of antianabolic steroids.[41,124,126,127,135] The

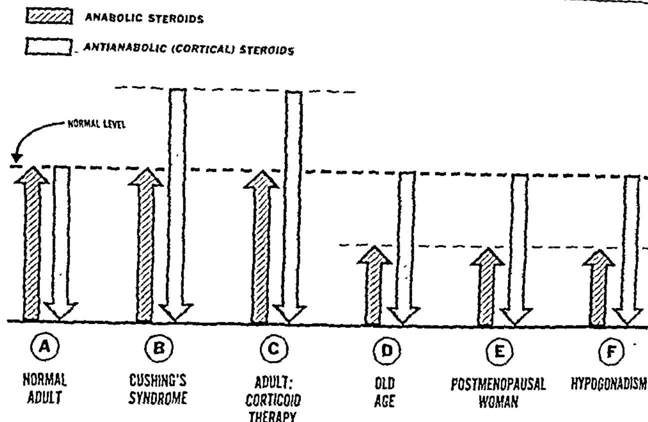


FIG. 25. Schematic diagrams of the balance between anabolic and antianabolic steroids in various physiologic and pathologic states.

An arrow pointing upward represents a stimulating effect; an arrow pointing downward represents an inhibiting effect; the length of an arrow indicates the amount of effect. The hatched arrows = anabolic steroids; the stippled arrows = cortical steroids. The heavy horizontal dash line = the normal level.

The relation between the endogenous levels of anabolic and antianabolic steroids is shown for six states: (A) the normal adult; (B) Cushing's syndrome; (C) the normal adult receiving chronic corticoid therapy; (D) old age; (E) the postmenopausal woman; and (F) hypogonadism with normal adrenal cortical function.

It will be seen that there is a balance between the hormonal influences in the normal adult (diagram A), that there is an absolute increase in the antianabolic action in Cushing's syndrome and in the normal adult receiving chronic corticoid therapy (diagrams B and C), and that there is a relative increase in the antianabolic action (as a result of a decrease in the anabolic action) in old age, in the postmenopausal woman and in hypogonadism with normal adrenal corticoid function (diagrams D, E & F).

(Modified from Reifenstein, E. C., Jr.: South. M. J. 49:933-960)

adverse effects of the relative excess of corticoid materials do not occur, however, presumably because the normal child also has a considerable production of anterior pituitary growth hormone, which is an anabolic agent.

Implications for the Development of Senile Osteoporosis

The alterations in hormonal balance previously discussed apparently take place in all persons as they age and, presumably, are adjustments that accompany the physiologic event that we term senescence. Since the hormonal imbalance is an important factor in the development of osteoporosis, some degree of senile osteoporosis should be present in all aging persons. This reasoning leads to the concept that osteoporosis is a physiologic process in aging bone.[8]

Investigators agree that *senile osteoporosis* is a very common condition. The question is, *does it occur in all old people?* It is most difficult to determine this because of the

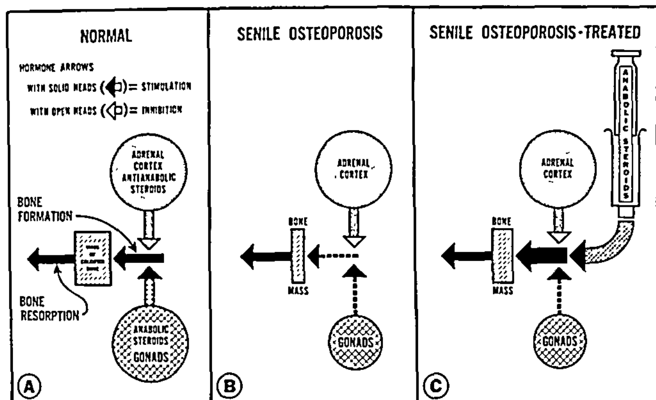


FIG. 26. Schematic diagrams illustrating the relationships of steroid hormones to the development and the treatment of senile osteoporosis.

In this chart the rectangle is the mass of calcified bone; the black arrows are the processes of bone formation and bone resorption, with the size of the arrows representing the rates of these processes; the circles are the adrenal cortex as a source of antianabolic hormones and the gonads as a source of anabolic steroids; the stippled arrows are the antianabolic steroids and the cross-hatched arrows the anabolic steroids, with the size of the arrows representing the amount of hormonal influence; the arrows with open heads are hormones with an inhibiting action, and those with solid heads are hormones with a stimulating action; the syringe labeled "anabolic steroids" represents administered anabolic therapy.

The chart has 3 divisions: (A) the normal status, in which the calcified mass of bone is in a state of dynamic equilibrium (because the rate of bone formation is equal to the rate of bone resorption) and the endogenous hormonal influences upon bone formation are in balance; (B) senile osteoporosis, in which the mass of calcified bone has decreased (because bone formation is reduced) and the endogenous hormonal influences upon bone formation are altered so that there is an absolute deficiency of anabolic steroids and a relative excess of antianabolic steroids; and (C) senile osteoporosis treated with anabolic steroid therapy, in which the calcified mass of bone is being increased toward normal by an increase in bone formation and the imbalance between the endogenous antianabolic and the anabolic hormones is being corrected by the administration of anabolic steroid therapy. For further discussion see text.

problems in detecting early or mild osteoporosis which we already have discussed. Furthermore, in old persons we encounter the additional difficulty of being uncertain as to what constitutes a "normal" roentgenographic bone-image density.

The fact remains that only a portion of the older population develops the skeletal deformities or presents the clinical manifestations of senile osteoporosis. This suggests that in certain individuals, additional factors have an important influence on the course of the osseous disorder. We do not know the nature of these factors. We can speculate that they may include such influences as the amount of calcium and phosphorus

in the skeleton at the time that bone formation decreases, the diet, the physical activity, the degree of alteration in the endogenous hormone levels, the development of other systemic derangements (such as vascular, renal, hepatic or acid-base disturbances), the occurrence of superimposed osseous disorders (such as atrophy of disuse, osteomalacia, fracture, arthritis or Paget's disease of bone) and such vague and nonspecific factors as individual variability and susceptibility. Difficulty in analyzing the role of these possible factors arises from the fact that in many cases osteoporosis of disuse, of malnutrition, of the postmenopausal state and of senility are inseparably superimposed. Furthermore, in some of the published studies, cases reported to have been osteoporosis appear from the data to have been cases of osteomalacia superimposed upon senile osteoporosis;[130] in others the patients appear to have had osteomalacia or other metabolic bone disorders rather than osteoporosis.[27]

Implications for the Treatment of Senile Osteoporosis

The obvious implication of the imbalance of the antianabolic and the anabolic steroids for the treatment of senile osteoporosis is to restore the balance through the administration of anabolic steroid medication The therapy should be given in adequate amounts and for a sufficient time to correct the protein depletion and osteoporosis.

In Figure 26 we have illustrated with schematic diagrams the relationships of steroid hormones to senile osteoporosis. The chart has three divisions: (A) the normal status, in which the calcified mass of bone is in a state of dynamic equilibrium because the rate of bone formation is equal to the rate of bone resorption and the endogenous hormonal influences upon bone formation are in balance; (B) senile osteoporosis, in which the mass of calcified bone has decreased because bone formation is reduced and the endogenous hormonal influences upon bone formation are altered so that there is an absolute deficiency of anabolic steroids and a relative excess of antianabolic steroids; and (C) senile osteoporosis treated with anabolic steroid therapy, in which the calcified mass of bone is being increased toward normal by an increase in bone formation and the imbalance between the endogenous antianabolic and the anabolic hormones is being corrected by the administration of anabolic steroid therapy. This figure summarizes the relationship of steroid hormones to the development of senile osteoporosis and the rationale for the use of anabolic steroid therapy in controlling the adverse effects of corticoid hormones upon osseous tissues.

Implications for the Prevention of Senile Osteoporosis

The evidence presented in this chapter provides strong support for the contention that anabolic steroids should be administered to all persons who have passed the prime of life and are becoming elderly, and particularly to all women at and after the time of the menopause, in order to prevent the development of protein depletion and osteoporosis. The dosage should be adjusted to maintain the patient in an optimal clinical state. Prolonged treatment will be required, since the underlying endogenous imbalance between anabolic and antianabolic steroids still is present whenever the anabolic steroid therapy is discontinued.

Practical Anabolic Steroid Therapy

At present the combination of androgen and estrogen provides the most potent and most practical anabolic preparation that is available for the control of senile osteoporosis. Such a combination has the advantages (1) of adding the more marked osseous-anabolic effect of the estrogen to the more marked protein-anabolic effect of the androgen and (2) of reducing the undesirable actions of either

hormone alone upon the genital organs and the accessory sexual structures. The preparation recently introduced by Junkmann and his colleagues,[40,59,60,133] in which 90 mg. of testosterone enanthate* and 4 mg. of estradiol valerate* are dissolved in each milliliter of vehicle, seems to meet very satisfactorily the requirements for anabolic steroid therapy. It has a potent anabolic effect (see Fig. 16) that is sustained for from 3 to 4 weeks after a single intramuscular injection of 1 to 2 ml., and it appears to have an optimal androgen-to-estrogen ratio that results in undesirable masculinizing or feminizing actions in only a very small percentage of patients of either sex. Senile osteoporosis may be treated, with less convenience to the patient or to the physician, by administering shorter-acting androgen-estrogen combinations at more frequent intervals by injection or daily by mouth. A few patients may develop moderate edema; this can be controlled by a low-sodium diet, diuretics and reduction of the steroid dosage. The last-mentioned step rarely is necessary.

In addition, the patients should (1) eat a high-protein diet to provide materials with which they can build the protein bone matrix; (2) take an adequate amount of water to avoid any tendency toward a concentrated urine when hypercalciuria is present; (3) avoid extra calcium or vitamin D during the initial phase of treatment when there is insufficient matrix to take up the mineral; (4) increase the calcium intake moderately after several months of steroid therapy to ensure calcification of all the matrix that is being formed; (5) begin a maximal amount of mobilization as soon as possible and avoid excessive immobilization to prevent adding atrophy of disuse to the bone condition; (6) wear a corset or a brace to support the mechanically defective areas; and (7) use their muscles so that strain of the weakened vertebral column is avoided.

* These steroid esters are available from E. R. Squibb & Sons, New York: testosterone enanthate as Delatestryl, estradiol valerate as Delestrogen; and the preparation mentioned above of testosterone enanthate combined with estradiol valerate as Deladumone.

SUMMARY AND CONCLUSIONS

Anabolic steroids (androgenic and estrogenic hormones of gonadal and adrenal cortical origin) stimulate the growth of protein and osseous tissues. Antianabolic steroids (corticoid hormones of adrenal cortical origin, such as hydrocortisone) inhibit the growth of protein and osseous tissues. In the presenile adult, these two opposing hormonal activities usually are in a state of balance. An endogenous imbalance can result in such individuals from either (1) an absolute increase in the amount of antianabolic steroids when the quantity of anabolic steroids is not changed, as in Cushing's syndrome or in chronic corticoid therapy, or (2) a relative increase in the amount of antianabolic steroids when the quantity of anabolic steroids is decreased, as in hypogonadism with normal adrenal cortical function. The imbalance is an important factor in the development of osteoporosis, a specific metabolic bone disease entity defined as that category of too little calcified bone in which there is too little formation of bone because of insufficient formation of matrix. The osteoporosis can be corrected by anabolic steroid therapy.

In the aging person there is an actual deficiency of endogenous anabolic hormones which creates at the same time a relative excess of endogenous antianabolic hormones. The imbalance qualitatively is the same as in Cushing's syndrome. The endogenous imbalance and particularly the relative excess of antianabolic hormones are important factors in the development of senile osteoporosis in the aging person. Again, the imbalance can be corrected by anabolic steroid therapy which replaces the missing quantity of anabolic steroids and counteracts the excessive quantity of antianabolic steroids. Some degree of senile

osteoporosis is present, but not recognizable, in all old people. Early stages or mild states of the osseous disturbance cannot be recognized at present because of the limitations in current diagnostic procedures. For indisputable roentgenographic evidence with existing technics, the bone calcium content must change at least 30 per cent, which usually takes from 5 to 10 years. These requirements apply also to evaluating in roentgenograms the initial response of senile osteoporosis to treatment. In addition to the hormonal imbalance, unknown factors influence the course of the osseous disorder in many aging individuals so that symptoms and deformities arise; when these manifestations appear, the diagnosis can be made with existing technics.

Anabolic steroids should be administered to restore and/or protect the protein and the osseous tissues in patients with recognizable senile osteoporosis, *in persons who are* becoming elderly, and particularly in women at and after the menopause. The combination of androgen and estrogen appears to be the most potent and the most practical anabolic preparation now available either for the treatment or for the prevention of senile osteoporosis.

REFERENCES

1. Albright, F.: Cushing's syndrome: its pathological physiology, its relationship to the adreno-genital syndrome, and its connection with the problem of the reaction of the body to injurious agents ("alarm reaction" of Selye), Harvey Lect. 38:123-186, 1942-1943
2. ———: The effect of hormones on osteogenesis in man *in* Recent Progress in Hormone Research (Proceedings of the Laurentian Hormone Conference), vol. 1, pp 293-353, 1947.
3. ———: Osteoporosis, Ann. Int. Med. 27: 861-882, 1947.
4. Albright, F., Bloomberg, E., and Smith, P. H.: Postmenopausal osteoporosis, Tr. A. Am. Physicians 55:298-305, 1940
5. Albright, F., Burnett, C. H., Cope, O., and Parson, W : Acute atrophy of bone (osteoporosis) simulating hyperparathyroidism, J. Clin. Endocrinol 1:711-716, 1941.
6. Albright, F., and Henneman, P. H.: Osteoporosis produced by cortisone-like steroids, Tr. Macy Conf. on Metabolic Interrelations, with Special Reference to Calcium 5:191-195, 1953.
7. Albright, F., Parson, W., and Bloomberg, E.: Cushing's syndrome interpreted as hyperadrenocorticism leading to hypergluconeogenesis: results of treatment with testosterone propionate, J. Clin. Endocrinol. 1:375-384, 1941.
8. Albright, F., and Reifenstein, E. C., Jr.: *The Parathyroid Glands and Metabolic* Bone Disease: Selected Studies, Baltimore, Williams & Wilkins, 1948.
9. Albright, F., Smith, P. H., and Fraser, R.: A syndrome characterized by primary ovarian insufficiency and decreased stature. Report of 11 cases with a digression on hormonal control of axillary and pubic hair. Am. J. M. Sc. 204:625-648, 1942.
10. Albright, F , Smith, P. H., and Richardson, A. M.: Postmenopausal osteoporosis: its clinical features, J.A.M.A. 116: 2465-2474, 1941.
11. Anderson, I. A.: Postmenopausal osteoporosis; clinical manifestations and the treatment with oestrogens, Quart. J. Med. 19:67-96, 1950.
12. Babaiantz, L.: The osteoporoses, *Radiol.* clin. 16:291-322, 1947.
13. Banghart, H. E.: A clinical evaluation of methyl androstenediol in the treatment of osteoporosis, Am. Pract. & Digest Treat 5:964-966, 1954.
14. Bartelheimer, H.: The clinical significance, the diagnosis, and the treatment of osteoporosis, Arztl. Wchnschr. 8:1137-1142, 1953.
15. Bartelheimer, H , and Schmitt-Rohde, J. M.. The Pathogenesis of Osteoporosis, Ergebn. inn. Med. u Kinderh. 7:454-585, 1956.
16. Bauer, W., and Bartter, F. C · Diseases of the bones *in* Cecil, R. L., and Loeb, R. F. (Eds.): Textbook of Medicine, ed. 9, pp. 1445-1475, Philadelphia, Saunders, 1955.
17. Berrios de la Luz, R , and Eulufi Marin, R.: Rheumatoid arthritis and Cushing's syndrome consecutive to prolonged use of cortisone; clinical case, Rev méd. Chile 81:565-568, 1953
18. Bishop, P. M. F., De Mowbray, R. R.,

Glover, F. N., and Thorne, M. G.: The treatment of Cushing's syndrome, Lancet 267:1137-1140, 1954.

19. Black, J. R., Ghormley, R. K., and Camp, J. D.: Senile osteoporosis of the spinal column, J.A.M.A. 117:2144-2150, 1941.
20. Bliss, E. A., Sandberg, A. A., Nelson, D. H., and Eik-Nes, K.: The normal levels of 17-hydroxycorticosteroids in the peripheral blood of man, J. Clin. Invest. 32:818-823, 1953.
21. Bogdonoff, M. D., Shock, N. W., and Parsons, J.: The effects of stilbestrol on the retention of nitrogen, calcium, phosphorus, and potassium in aged males with and without osteoporosis, J. Gerontol. 9: 262-275, 1954.
22. Boland, E. W., and Headley, N. E.: Management of rheumatoid arthritis with smaller (maintenance) doses of cortisone acetate, J.A.M.A. 144:365-372, 1950.
23. Bunim, J. J., Ziff, M., and McEwen, C.: Evaluation of prolonged cortisone therapy in rheumatoid arthritis: a four-year study, Am. J. Med. 18:27-40, 1955.
24. Burrage, W. S. *et al.*: The use of cortisone and hydrocortisone in allergy, Ann. Int. Med. 43:1001-1018, 1955.
25. Case Records of the Massachusetts General Hospital: Case 39221, New England J. Med. 248:945-950, 1953.
26. Cooke, R. M.: Osteoporosis, Lancet 268: 877-882, 929-937, 1955.
27. Cooke, W. T., Barclay, J. A., Govan, A. D. T., and Nagley, L.: Osteoporosis associated with low serum phosphorus and renal glycosuria, A.M.A. Arch. Int. Med. 80:147-164, 1947.
28. Crispell, K. R., Parson, W., and Harden, G.: The relation of dietary protein consumption to N-15 excretion in normal subjects and in Cushing's syndrome utilizing N-15 glycine orally and intravenously, J. Clin. Invest. 33:342-346, 1954.
29. Curtiss, P. H., Jr., Clark, W. S., and Herndon, C. H.: Vertebral fractures resulting from prolonged cortisone and corticotropin therapy, J.A.M.A. 156:467-469, 1954.
30. Deakins, M. L., Friedgood, H. B., and Ferrebee, J. W.: Some effects of testosterone, testosterone propionate, methyltestosterone, stibestrol, and x-ray therapy in a patient with Cushing's syndrome, J. Clin. Endocrinol. 7:376-384, 1947.
31. Del Castillo, E. B., De La Balze, F. A., and Argonz, J.: Syndrome of rudimentary ovaries with estrogenic insufficiency and increase in gonadotropins, J. Clin. Endocrinol. 7:385-422, 1947.
32. Demartini, F., Grokoest, A. W., and Ragan, C.: Pathological fractures in patients with rheumatoid arthritis treated with cortisone, J.A.M.A. 149:750-752, 1952.
33. de Sèze, S., Hubalt, A., and Renier, J. C.: Four cases of spontaneous fractures observed in disorders treated with cortisone, French League Against Rheumatism, Meeting Jan. 14, 1953; Presse méd. 61: 819, 1953.
34. Dunn, C. W.: The Cushing syndrome, Endocrinol. 22:374-385, 1938.
35. ———: Oestrin in the treatment of Cushing's syndrome, Lancet 233:344, 828, 1937.
36. Eisenhardt, L., and Thompson, K. W.: Brief consideration of the present status of so-called pituitary basophilism, with tabulation of verified cases, Yale J. Biol. & Med. 11:507-522, 1939.
37. Eisenstadt, W. S., and Cohen, E. B.: Osteoporosis and compression fractures from prolonged cortisone and corticotropin therapy, Ann. Allergy 13:252-256, 1955.
38. Eliel, L. P., Hellman, L., Pearson, O. H., and Katz, B.: The effects of ACTH on the electrolyte content of various body tissues *in* Mote, J. R. (Ed.): Proceedings of the Second Clinical ACTH Conference, vol. 1, pp. 196-212, New York, Blakiston, 1951.
39. Engleman, E. P., *et al.*: Rheumatoid arthritis: an evaluation of long-term treatment with cortisone, California Med. 80: 369-374, 1954.
40. Frank, E., Froewis, J., Rockenschaub, A., and Ulm, R.: Combined treatment of ovarian insufficiency phenomena with estrogenic and androgenic steroid hormones in a depot form, Zentralbl. Gynak. 76:859-861, 1954.
41. Fraser, R. W., *et al.*: Colorimetric assay of 17-ketosteroids in urine. A survey of the use of this test in endocrine investigation, diagnosis, and therapy. J. Clin. Endocrinol 1:234-256, 1941.
42. Fusi, G.: Experimental study on the first radiological manifestation of osteoporosis, Radiol. clin. 22:123-129, 1953.

43. Gershon-Cohen, J., Rechtman, A. M., and Schraer, H.: Asymptomatic fractures in osteoporotic spines of the aged, J.A.M.A. 153:625-627, 1953.
44. Gill, A. M.: Treatment of Cushing's syndrome with large doses of oestrin, Lancet 233:70-72, 1937.
45. Gordan, G. S.: Anabolic Therapy. Proceedings of a Conference on The Clinical Use of Anabolic Agents, Chicago, Searle Research Laboratories, 1956.
46. ———: Evaluation and use of anabolic steroids, GP 10:87-102, 1954.
47. ———: Metabolic bone diseases in practice: osteoporosis, Paget's disease, von Recklinghausen's disease, and Albright's syndrome, Am. Pract. & Digest Treat. 2: 113-119, 1951.
48. Greenblatt, R. B., and Nieburgs, H. E.: Turner's syndrome, J. Clin. Endocrinol. 8:993-1003, 1948.
49. Greenlee, R. G.: Hyperfunction of the adrenal cortex, M Clin. North America 39:1081-1099, 1955.
50. Greisheimer, E. M.: Postmenopausal osteoporosis, J. Am. M. Women's A. 6: 183-184, 1951.
51. Hamburger, C.: Normal urinary excretion of neutral 17-ketosteroids with special reference to age and sex variations, Acta endocrinol. 1:19-37, 1948.
52. Hamilton, H. B., and Hamilton, J. B.: Aging in apparently normal men. I. Urinary titers of ketosteroids and of alpha-hydroxy and beta-hydroxy ketosteroids J. Clin. Endocrinol. 8:433-452, 1948.
53. Hart, G. M.: Postmenopausal osteoporosis of the spine, Geriatrics 5:321-330, 1950.
54. Howard, R. P.: Medical aspects of bone disease with particular reference to osteoporosis, Canad. M. A J 63:258-264, 1950.
55. Howard, R P., and Reifenstein, E. C., Jr.. Evidence that the long-acting steroid, testosterone enanthate, is more effective than the short-acting steroids, testosterone propionate and methyltestosterone, in stimulating protein anabolism in osteoporosis (postmenopausal and senile) In press
56. Hubble, D : Cushing's syndrome and thymic carcinoma, Quart. J Med. 18:133-147, 1949.
57. Irwin, J. W , Henneman, P. H., Wang, D M. K., and Burrage, W. S.: Maintenance cortisone in intractable asthma: preliminary observations of undesirable cortisone effects, J. Allergy 25:201-209, 1954.
58. Jores, A.: The genital organs and their diseases in Handbuch der inneren Medizin, ed. 4, vol. 7, Berlin, Springer, 1955.
59. Junkmann, K.: Long-acting androgens, Arch. exper. Path. u. Pharmakol. 215:85-92, 1952.
60. ———: Long-acting estrogens, Arch. exper. Path. u. Pharmakol. 220:358-364, 1953.
61. Kepler, E. J., Sprague, R. G., Mason, H. L., and Power, M. H.: Aspects of clinical endocrinology: the pathologic [illegible] Laurentian Hormone Conference), vol. 2, pp. 345-389, 1948.
62. Kinsell, L. W.: ACTH and cortisone—after nearly 4 years of intensive clinical and experimental evaluation, J. Clin Endocrinol. 13:120-122, 1953.
63. ———:Consideration of calcium and phosphorus metabolism, including the normal and pathologic physiology of the parathyroid glands, Am. Pract. & Digest Treat 3:499-505, 1949
64. Kinsell, L W , Boling, L., Partridge, J. W., and Foreman, N.: Corticotropin (ACTH) and cortisone; newer concepts of their use in clinical practice, California Med. 78:487-490, 1953.
65. Knowlton, A. I.: Cushing's syndrome, Bull. New York Acad Med. 29:441-465, 1953.
66. Kochakian, C. D The protein anabolic effects of steroid hormones in Harris, R. S., and Thimann, K. V. (Eds.): Vitamins and Hormones, vol. 4, pp 255-310, New York, Acad. Press, 1946
67. Kolb, F. O : Metabolic diseases of the bone in the adult, Kaiser Foundation M. Bull 4:348-360, 1956.
68 Labhart, A., and Courvoisier, B.: Osteoporosis in a case of eunuchism, Helvet. med. acta 17:475-479, 1950
69. Labhart, A., and Schüpbach, A : Treatment of osteoporosis with sex hormones, Schweiz med. Wchnschr. 81:992-995, 1951
70 Lachman, E.: Osteoporosis. the potentialities and limitations of its roentgenologic

diagnosis, Am. J. Roentgenol. 74:712-715, 1955.

71. Lachman, E., and Whelan, M.: Roentgen diagnosis of osteoporosis and its limitations, Radiology 26:165-177, 1936.

72. Laszlo, D., and Spencer, H.: Newer techniques in the study of calcium metabolism in man and effects of hormones thereon *in* Engle, E. T., and Pincus, G (Eds.): Hormones and the Aging Process, pp. 175-200, New York, Acad. Press, 1956.

73. Lichtwitz, A , and Clément, D.: The humoral and tissular regulation of calcium metabolism, Semaine hôp., Paris 30:2911-2922, 1954.

74 Lichtwitz, A., Parlier, R., Clément, D., and Delaville, M.: The metabolism of calcium and steroids, Semaine hôp. Paris 31: 554-562, 1955.

75. Luder, J.: Vertebral collapse after cortisone therapy, London, Great Ormond St. J. 7:15-21, 1954.

76. MacKenzie, D. A., and Janes, J. M.: Postmenopausal osteoporosis: a programme of treatment in 42 Cases, Canad. M. A. J. 71:339-340, 1954.

77. Michon, P., Lochard (Mme), and Gaussin: Implantation of estradiol (estrogen) in postmenopausal osteoporosis; report of a case, Rev. méd. Nancy 73:143-147, 1948.

78. Moldawer, M., Pathogenesis and treatment of senile osteoporosis, Geriatrics 11:319-331, 1956.

79. ———. Senile osteoporosis: the physiological basis of treatment, A.M.A Arch Int. Med 96:202-214, 1955.

80. Nowakowski, H : The Action of Sex Hormones on the Skeleton and Its Metabolism, The Metabolic Action of Steroid Hormones, pp. 93-110, Berlin, Springer, 1955.

81. Nowakowski, H., and Gadermann, E.: Regressive vertebral column changes with bilateral testicular atrophy and anorchism, Verhandl. deutsch. Gesellsch. inn. Med. 58:400-405, 1952.

82. Parson, W., Crispell, K. R , and Ebbert, A , Jr.: Abnormalities in N^{15} excretion rates after ingestion of tagged glycine in Cushing's syndrome and following ACTH administration, J. Clin. Invest 31:548-554, 1952.

83. Paschkis, K. E , Herbut, P. A., Rakoff, A E., and Cantarow, A.: A case of Cushing's syndrome with adrenal cortical hyperplasia, without pituitary basophilic adenoma or hyperplasia, J. Clin. Endocrinol. 3:212-217, 1943.

84. Paschkis, K. E., Rakoff, A. E., and Cantarow, A.: Clinical Endocrinology, pp. 552-553, New York, Hoeber, 1954.

85. Pearson, O. H., and Eliel, L. P.: Hormone therapy in cancer: experimental studies with ACTH and cortisone in patients with neoplastic disease *in* Recent Progress in Hormone Research (Proceedings of the Laurentian Hormone Conference), vol. 6, pp. 373-416, 1951.

86. Pearson, S., Weissberg, J., and McGavack, T. H.: Steroid studies. I. Metabolic effects of androstanolone in aged people. J. Am. Geriatrics Soc. 2:26-31, 1954.

87. Perloff, W. H., Boutwell, J. H., Jr., and Maas, R.: The endocrine treatment of climacteric (steroid-deficiency) osteoporosis, J. Am. Geriatrics Soc. 4:760-765, 1956.

88 Perloff, W. H., Rose, E., and Sunderman, F. W.: Therapeutic observations in Cushing's syndrome: effect of various agents on calcium, phosphorus, and nitrogen excretion in a patient with pituitary basophilism, A.M.A. Arch. Int. Med. 72:494-505, 1943.

89. Philipp, E.: Congenital absence of the genital organs, Geburtsh. u. Frauenh. 11:193-206, 1951.

90. Pincus, G.: Aging and urinary steroid excretion *in* Engle, E. T., and Pincus, G. (Eds.): Hormones and the Aging Process, pp 1-20, New York, Acad. Press, 1956.

91. Pincus, G., Dorfman, R. I , *et al* : Steroid metabolism in aging men and women *in* Recent Progress in Hormone Research (Proceedings of the Laurentian Hormone Conference), vol. 11, pp. 307-341, 1955.

92. Pincus, G., Romanoff, L. P., and Carlo, J.: The excretion of urinary steroids by men and women of various ages, J. Gerontol. 9:113-132, 1954.

93. Rakoff, A. E., Cantarow, A., and Paschkis, K. E.: Cushing's syndrome (two cases treated with stilbestrol), J. Clin Endocrinol. 1:912-915, 1941.

94. Reifenstein, E. C., Jr.: The definitions, terminology and classification of metabolic bone disorders *in* DePalma, A. F. (Ed.): Clinical Orthopaedics No. 9, pp. 30-45, Philadelphia, Lippincott, 1957.

95. ———: Diseases of the parathyroid glands *in* Williams, R. H. (Ed.): Textbook of Endocrinology, ed. 2, pp. 483-581, 717-727, Philadelphia, Saunders, 1955.
96. ———: Metabolic disorders of bone *in* Harrison, T. R. (Ed.): Principles of Internal Medicine, ed. 2, pp. 404-418, 697-730, New York, Blakiston, 1954.
97. ———: The rationale for the use of anabolic steroids in controlling the adverse effects of corticoid hormones upon protein and osseous tissues, South. M. J. 49:933-960, 1956.
98. Reifenstein, E. C., Jr., and Albright, F.: The metabolic effects of steroid hormones in osteoporosis, J. Clin. Invest. 26:24-56, 1947.
99. Reifenstein, E. C., Jr., Albright, F., and Wells, S. L.: The accumulation, interpretation, and presentation of data pertaining to metabolic balances, notably those of calcium, phosphorus, and nitrogen, J. Clin. Endocrinol. 5:367-395, 1945.
100. Reifenstein, E. C., Jr., and Howard, R. P.: The parathyroid glands *in* Glandular Physiology and Therapy, chap. 51, pp. 351-385 (published under the auspices of the Council on Pharmacy and Chemistry, A.M.A.), Philadelphia, Lippincott, 1954.
101. Robinson, W. D., *et al.*: Rheumatism and arthritis. Review of American and English literature of recent years (Tenth Rheumatism Review). Ann. Int. Med. 39:498-618, 757-906, 1953.
102. Roth, K.: Combination hormone treatment of dystrophia adiposagenitalis with hypothyroidism and severe osteoporosis, Ther. Gegenw. 93:433, 1954.
103. Samuels, L. T.: Effect of aging on the steroid metabolism as reflected in plasma levels *in* Engle, E. T., and Pincus, G. (Eds.): Hormones and the Aging Process, pp 21-38, New York, Acad. Press, 1956.
104. Schoene, R. H: A clinical approach to senile osteoporosis, Ohio M. J. 48:126-129, 1952.
105. Sherman, M. S.: Estrogens and bone formation in the human female, J. Bone & Joint Surg. 30A:915-930, 1948.
106. Shohl, A. T.: Mineral Metabolism, p. 19, New York, Reinhold, 1939.
107. Shorr, E.: Effect of strontium on the calcium balance of patients with osteoporosis, Tr. Macy Conf. Metabolic Aspects of Convalescence, Including Bone & Wound Healing 6:125-129, 1944.
108. Shorr, E., and Carter, A. C.: Strontium as an adjuvant in the treatment of postmenopausal osteoporosis, Tr. Macy Conf. Metabolic Aspects of Convalescence, Including Bone & Wound Healing 16:122-146, 1947.
109. ———: Studies on the effect of estrogens, androgens, and vitamin D_2 on the calcium and the strontium metabolism, Tr. Macy Conf. Metabolic Aspects of Convalescence, Including Bone & Wound Healing 15:99-113, 1947.
110. ———: The usefulness of strontium as an adjuvant to calcium in the remineralization of the skeleton in man, Bull Hosp. Joint Dis. 13:59-66, 1952.
111. ———: The value of strontium as an adjuvant to calcium in the remineralization of the skeleton in osteoporosis in man, Tr. Macy Conf. Metabolic Interrelations, with Special Reference to Calcium 2:144-154, 1950.
112. Snapper, I.: Osteoporosis, M. Clin. North America 36:847-863, 1952.
113. Sobel, H., and Marmorston, J.: The effect of cortisone on the collagen and hexosamine content of the skin and femurs of one-year-old rats, Endocrinology 55:21-27, 1954.
114. ———: The possible role of the gel-fiber ratio of connective tissue in the aging process, J. Gerontol. 11:2-7, 1956.
115. Sobel, H., Marmorston, J., and Moore, F. J.: Collagen and hexosamine content of femurs of rats, Proc. Soc. Exper. Biol. & Med. 87:346-349, 1954.
116. Sobel, H., Zutrauen, H. A., and Marmorston, J.: The collagen and hexosamine content of the skin of normal and experimentally treated rats, Arch Biochem. 46:221-231, 1953.
117. Soffer, L. J., and Bader, R.: Corticotropin and cortisone in acute disseminated lupus erythematosus. Results of long-term use, J.A.M.A. 149:1002-1008, 1952.
118. Soffer, L. J., Elster, S. K., and Hamerman, D. J.: Treatment of acute disseminated lupus erythematosus with corticotropin and cortisone, A M.A. Arch. Int Med. 93:503-514, 1954.
119. Sohval, A. R., and Soffer, L. J.: Congenital testicular deficiency. I. Absence of

spermatogonia, Sertoli and Leydig cells as cause of eunuchoidism with cryptorchidism, J. Clin. Endocrinol. 12:1229-1238, 1952.

120. Spencer, H., Hausinger, A., and Laszlo, D.: The calcium tolerance test in senile osteoporosis, J. Am. Geriatrics Soc. 2:19-25, 1954.

121. Sprague, R. G., Mason, H. L., and Power, M. H.: Chemistry, physiology, and clinical endocrinology of the adrenal cortex: physiologic effects of cortisone and ACTH in man *in* Recent Progress in Hormone Research (Proceedings of the Laurentian Hormone Conference), vol. 6, pp. 315-372, 1951.

122. Sprague, R. G., Power, M. H., *et al.*: Observations on the physiologic effects of cortisone and ACTH in man, A. M. A. Arch. Int. Med 85:199-258, 1950.

123. Steinbrocker, O., *et al.*: The clinical application of cortisone and ACTH in arthritis and related conditions: methods and problems. II. Side-effects, complications, contraindications, precautions and conclusions. Arizona Med. 8:2935, 1951.

124. Talbot, N. B., and Butler, A. M.: Urinary 17-ketosteroid assays in clinical medicine, J. Clin. Endocrinol. 2:724-729, 1942.

125. Talbot, N. B., Sobel, E. H., *et al.*: Functional Endocrinology: From Birth Through Adolescence, p. 227, Cambridge, Mass., Harvard, 1952.

126. Talbot, N. B., Wood, M. S., *et al*: Further observations on the urinary excretion of water-soluble corticosteroids by normal and abnormal subjects, J. Clin. Endocrinol. 11:1224-1236, 1951.

127. Talbot, N B., Zygmuntowicz, A. S., *et al.*: Observations on adrenal cortical "sugar-fat-nitrogen" hormone ("11-17-OCS") and "17-ketosteroid precursor" production by normal and abnormal individuals of various ages with comments on the fact that (a) there may be two ACTH's and (b) the normal adrenal cortex may not produce true androgens *in* Mote, J. R. (Ed.): Proceedings of the First Clinical ACTH Conference, chap. 4, pp. 32-37, Philadelphia, Blakiston, 1950.

128. Teabeaut, R., Engel, F. L., and Taylor, H.: Hypokaliemic, hypochloremic alkalosis in Cushing's syndrome. Observations on the effects of treatment with potassium chloride and testosterone. J. Clin. Endocrinol. 10:399-409, 1950.

129. Teicher, R., and Nelson, C. T.: Osteoporosis and pathological fractures following treatment with ACTH and cortisone, J. Invest. Dermat. 19:205-210, 1952.

130. Thorn, G. W., *et al.*: Pharmacologic aspects of adrenocortical steroids and ACTH in man, New England J. Med. 248:232-245, 284-294, 323-337, 369-378, 414-423 and 588-601, 1953.

131. ———: Clinical uses of ACTH, cortisone, and hydrocortisone: specific replacement therapy, New England J. Med. 248:632-646, 1953.

132. Tutt, M., Kidman, B., Rayner, B., and Vaughan, J.: The deposition of Sr_{89} in rabbit bones following intravenous injection, Brit. J. Exper. Path. 33:207-215, 1952.

133. Ufer, J.: Treatment of climacteric symptoms with long-acting hormone ester mixtures, Geburtsh. u. Frauenh. 14:650-655, 1954.

134. Varney, R. F., Kenyon, A. T., and Koch, F. C.: An association of short stature, retarded sexual development and high urinary gonadotropin titers in women, J. Clin. Endocrinol. 2:137-145, 1942.

135. Venning, E. H., and Browne, J. S. L.: Excretion of glycogenic corticoids and of 17-ketosteroids in various endocrine and other disorders, J. Clin. Endocrinol. 7: 79-101, 1947.

136. Vinther-Paulsen, N.: Calcium and phosphorus intake in senile osteoporosis, Geriatrics 8:76-79, 1953.

137. Watkin, D. M., Parsons, J. M., Yiengst, M. J., and Shock, N. W.: Metabolism in the aged: the effect of stanolone on the retention of nitrogen, potassium, phosphorus, and calcium and on the urinary excretion of 17-keto, 11-oxy, and 17-hydroxy steroids in 8 elderly men on high and low protein diets, J. Gerontol. 10: 268-287, 1955.

138. Weinberg, M.: Osteoporosis: diagnosis and treatment, J. Am. Geriatrics Soc. 4:429-437, 1956.

139. Welch, R. G., and Forsyth, C. C.: Still's disease treated with ACTH and cortisone, Great Ormond St. J. 5:1-6, 1953.

140. West, H. F., and Newns, G. R.: Cortisone and rheumatoid disease, Lancet 265: 1123-1126, 1953.

141. Whedon, G. D.: Steroid hormones in osteoporosis *in* Engle, E. T., and Pincus, G. (Eds.): Hormones and the Aging Process, pp. 221-239, New York, Acad. Press, 1956.

142. Whitelaw, M. J.: A case of Cushing's syndrome treated with testosterone propionate, J. Clin. Endocrinol. 4:480-482, 1944.

143. Wilkins, L.: A feminizing adrenal tumor causing gynecomastia in a boy of 5 years contrasted with a virilizing tumor in a 5-year-old girl; classification of 70 cases of adrenal tumor in children according to their hormonal manifestations and a review of 11 cases of feminizing adrenal tumor in adults, J. Clin. Endocrinol. 8: 111-132, 1948.

144. Wolf, J., and Loeser, A. A.: Testosterone therapy for fractures and certain malignant and nonmalignant bone conditions, J. Clin. Endocrinol. 14:107-109, 1954.

Le Relationes Inter le Hormones Steroide e le Disveloppamento e le Tractamento de Osteoporosis in Personas de Etate Avantiate

Summario in Interlingua

Steroides anabolic (hormones andro- e estrogene de origine gonadal e adrenocortical) stimula le crescentia de tessuto proteinic e ossee. Steroides anti-anabolic (hormones corticoide de origine adrenocortical, como per exemplo hydrocortisona) inhibi le crescentia de tessuto proteinic e ossee In adultos presenil, iste activitates hormonal contrari es usualmente in stato de equilibrio. Disequilibrio endogene pote resultar, in tal individuos, (1) ab un augmento absolute del steroides anti-anabolic sin alteration del quantitate del steroides anabolic, per exemplo in syndrome de Cushing o in chronic therapias corticoide, o (2) ab un augmento relative del steroides anti-anabolic in consequentia del reduction del quantitate

In personas de etate avantiate, il existe de facto un carentia de endogene hormones anabolic, lo que resulta in un excesso relative de endogene hormones anti-anabolic. Le disequilibrio es qualitativemente le mesme como in casos de syndrome de Cushing Le disequilibrio—e specificamente le excesso relative de hormones anti-anabolic—es un del factores importante in le disveloppamento de osteoporosis senil in personas de etate avantiate. Como previemente dicite, iste disequilibrio pote esser corrigite per therapias a steroide anabolic que reimplacia le manco de steroides anabolic e contraria le excesso de steroides anti-anabolic. Un certe grado de osteoporosis senil es presente, ben que non [illegible], in omne personas de

evidentia roentgenographic del prime responsa de osteoporosis senil al effecto del tractamento. A parte le disequilibrio hormonal, varie non-cognoscite factores exerce un influentia super le curso del disordine ossee in multe personas de etate avantiate, con le resultado del declaration de clar symptomas e deformitates. In le presentia de tal manifestationes, le diagnose pote esser establite in despecto del limitationes del technicas nunc disponibile.

Steroides anabolic deberea esser administrate pro restaurar e/o proteger le tessutos proteinic e ossee in patientes con recognoscibile osteoporosis senil, in personas qui attinge un etate avantiate, e specialmente in feminas al menopause e postea. Le combination de androgeno con estrogeno es apparentemente le plus potente e le plus practic preparato anabolic nunc disponibile pro le tractamento e etiam pro le prevention de osteoporosis senil.

Section III

GENERAL ORTHOPAEDICS

20

Another Approach to the Treatment of Spondylolisthesis and Spondyloschisis

ARTHUR B. KING, M.D., D. R. BAKER, M.D., AND W. J. MCHOLICK*

INTRODUCTION

Among the causes of low back pain and/or sciatica is the condition known as spondylolisthesis. It has long been recognized that there exists in this disorder a dehiscence in the bony arch of the neural canal of the vertebra with separation of the body anteriorly and the arch posteriorly. With the advent of more punctilious scrutiny of the lower lumbar region by means of good-quality roentgenograms, it is being noted with increasing frequency that defects in the neural arch may occur without displacement of either fragment. The term *spondyloschisis* is used to define this latter condition, hence the term *spondylolisthesis* would appear to be a matter of degree, indicating that a separation of the parts has come about.

Before embarking into a detailed discussion of the subject of treatment, it will be well to pause for a moment to consider the incidence and the symptomatology of this bony anomaly. Since the advent of pre-employment roentgenographic study of the lumbosacral spine, it is becoming clear that both these conditions exist in much greater number than was previously supposed. It is also evident that often people who harbor such lesions are asymptomatic, so that their mere presence does not mean a priori an unstable back with its concomitant symptoms. We feel strongly that this point should be recognized clearly by the physician before he makes a gloomy prediction for the patient and recommends drastic therapy. We believe that the asymptomatic patient with either spondylolisthesis or spondyloschisis should be left alone and not restricted in activity. We have been unable to uncover any detailed convincing evidence, either in the literature or in our own experience, that indicates that limited physical exertion or the conservative measures advocated in the past have resulted in a long-term relief of symptoms in a significant percentage of patients, nor have they prevented the onset of complaints in the previously asymptomatic patient.

BACKGROUND AND TERMINOLOGY

The disorder of the spinal column known as spondylolisthesis has been clearly defined by past authors as the anterior displacement of the body of one vertebra on another, associated with a bilateral defect in the region of the pars interarticularis, allowing the posterior portion of the neural arch to remain in position or to be displaced backward. Credit for the original use of the term *spondylolisthesis* is given to Kilian[16] who, in 1853, described such a case, although there are reports of earlier recognition of the disorder.

It was not until 1892 that Neugebauer[20] reviewed 101 cases and produced the first

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accurate conception of the essential pathology. He recognized that there was a defect in the pars interarticularis and, in fact, used the term *spondyloschisis interarticularis*. The obstetricians had long been aware of the disorder and its encroachment into the pelvic inlet. Thus, prior to 1900, all but six of the reported 125 cases were females, producing a false impression as to its age incidence and sex distribution.

The term *spondyloschisis* is used to identify those cases in which there is a defect in the continuity of the pars interarticularis *without displacement* of the vertebral body. This condition was not recognized clinically until 1924 when Whitman[17,23] reported five such cases before the American Orthopedic Association. In a study of skeletons of American aborigines, Congdon pointed out the great frequency of spondyloschisis and the extreme difficulty of trying to demonstrate the defect by x-ray.[9] It is still poorly understood, and frequently it is undiagnosed in the conventional roentgenographic examination of the low back.

Besides prespondylolisthesis, the term *spondylolysis* has been used synonymously with spondyloschisis. We object to the use of the former term because it implies that the vertebral body is freely movable and that there will be an inevitable slippage, while the latter term infers dissolution of an arch that previously was intact. While we grant that at some time during life a patient with spondylolisthesis must have been in the stage of spondyloschisis, we aver strongly that the former is not the inevitable end-result or, even, that it occurs frequently. We have encountered several patients who have had pronounced clinical symptoms for from 25 to 30 years because of spondyloschisis in whom no slip of the vertebral body has occurred. There are exceedingly few references to definitely proved cases of progressive slip of the vertebral body despite the alarm generated by most orthopaedic surgeons over its possible occurrence.

Hitchcock[14] pointed out that prior to Friberg's report,[11] no cases of spondylolisthesis had slipped further than that noted on the original diagnosis. He pointed out that if the assumed further slip did occur, it was very slow and gradual, and this has been our own experience. Two of our patients have had a slip of 3 or 4 mm. over a period of several years. At operation a uniform finding has been a vertebral body that is firmly fixed and often appears to be fused.

No patients with so-called retrospondylolisthesis have been recognized in our series. Pseudospondylolisthesis is a condition that is not germane to the present discussion.

ETIOLOGY

As yet there is no meeting of minds as to the cause of the dehiscence in the pars interarticularis. Early investigators assumed that the defects were secondary to inflammation. No convincing evidence has been brought forward to support this contention.

That trauma was the underlying cause has long been an attractive and, presumably, an obvious cause. The difficulty has been to gather specific facts. First, to our knowledge no patient has been reported who had absolutely normal roentgenographic findings preceding trauma and later was demonstrated to have such a defect. Second, there is the total absence of the usual signs of healing. At no time have we, or others, seen callus or new bone formation about the "break" on roentgenographic study at operation or at autopsy. This is most peculiar and is at variance with ordinary acute fractures of the vertebral body or the neural arch which almost invariably show vigorous new bone formation.

Many specimens removed by us at operation have been examined, and the region of defect always is uniform in its appearance. The bone merges gradually into fibrous tissue. This has been confirmed microscopically. Sharp or eburnated bony ends characteristic of an ununited fracture have not been seen. "Spurs" or "knobs" are frequent, which strike us as abortive and misdirected development and not attempts at natural healing. The pars interarticularis on either

side varies tremendously in length. Often it points in directions that never would meet the superior facet. In more than half our patients, there were additional free pieces of bone lying in the ligamentous attachments between the facets. Some of these were beneath the level of the superior facets.

One of us (ABK) operated upon a patient who had sustained a pronounced flexion injury when thrown out of a speeding automobile. The L-1 vertebra was crushed and dislocated posteriorly on T-12. The intervertebral disk was extruded, and the interspinous ligaments and the ligamentum flavum were ruptured. Both pedicles of L-1 were snapped off, and, in addition, both pars interarticularis were sheared off at the inferior facets. The conus of the spinal cord was pulverized. It was apparent to us that nothing short of such violence could produce such a break in the pars interarticularis in the adult. None of the patients whom we have encountered with spondylolisthesis and spondyloschisis gives any history of trauma that approaches such violence. Many, however, do give a history of a fall, a twist or a bend that produced a "snap" in the low back. Often acute symptoms date from this time, but usually one can get a history of low back discomfort preceding this if one persists in interviewing close relatives as well as the patient and no legal liability is involved. It is our contention that some of the ligamentous attachments of the free arch are stretched or disrupted by trauma, which allows the arch to become more mobile. This permits impingement on the underlying nerve roots and hypermobility at the inferior facets, all of which is painful.

To avoid the stumbling block of violent trauma needed to fracture the pars interarticularis in adults, many authors have postulated abnormal strains in infancy and in childhood. While much has been written on this point, very little in the way of concrete evidence has been presented.

That the defect is congenital is the theory with most support at the present time. This, too, is far from being on a firm basis. Recent detailed pre-employment roentgenographic examinations have revealed that about 50 per cent of "normal" people have bony anomalies about the lumbosacral area. Meyerding[10] stated that between 70 and 100 per cent of the reported cases of spondylolisthesis had associated anomalies. This tends to the belief that such defects could easily occur in this region. Just why the lesion should occur is not explained. Absence or duplication of ossification centers or failure of fusion of centers has been postulated, but laboratory evidence is missing.

Batts[4] has made an observation that while the incidence of spondylolisthesis in adults is about 5 per cent, he was unable to find anomalous ossification centers in the skeletons of 200 infants. It has been the failure to find the defective neural arches in infants that has lead to the speculation that trauma in very early life is the cause. This idea has been espoused by Hitchcock,[14] who found that he could produce such a defect in the neural arch by hyperflexion, with moderate force, of the spine of an infant cadaver. Again, evidence of healing has been conspicuous by its absence, nor has proof been offered that such fractures occur during early life.

To complicate matters further, George[12] and Friberg[11] have reported instances in which the defect appeared in abnormal frequency in several families. One is left, then, with several conflicting theories regarding its cause. We lean toward the belief that the defect is congenital. At all events, while this trouble is present from very early life, it manifests itself generally many years later and often not at all. The youngest patient with typical roentgenographic findings of spondylolisthesis of whom we have heard was reported by Kleinberg[18]—an infant of 17 months.

Two of us (DRB & WJM) have surveyed 400 schoolchildren in the first grade. We found eleven instances of spondylolisthesis



FIG. 1. Section taken through the end of the pars interarticularis and extending into the attached fibrous band. The gradual transition from cartilaginous material to dense fibrous tissue is evident. There is nothing to suggest an ununited fracture or callus formation.



FIG. 2. Section taken a little farther posteriorly, showing the transition from bone to cartilage. No evidence of trauma or infection is seen.

and seven cases of unilateral defects of the pars interarticularis, but no instances of bilateral spondyloschisis, all asymptomatic. This latter fact is most difficult to explain in view of the 30 per cent incidence of spondyloschisis found at surgery in our adult cases. It is possible that some of the seven unilateral cases may be shown eventually to be bilateral. This may be due to our present inability to obtain clear roentgenographic definition of the bony structure of the neural arch. Thus, the normal "adult" rate of about 5 per cent is present by the sixth year of life Similar lesions were found in the families of 17 affected children in a much larger percentage than in the normal population.[3]

In an effort to determine the incidence of neural arch defects in the population, several excellent papers based on anatomic studies are available. Those of Roche and of Congdon should be consulted.

PATHOLOGY

With the exception of one case, our experience surgically has been limited to bilateral defects in the pars interarticularis. Of the cases subjected to operation, the arch has been defective at L-5 in 27 cases and at L-4 in 2 cases. A single patient with a unilateral defect at L-3 who developed sufficient rotation of the pars interarticularis to press upon the L-4 root has been operated upon by us. This case is similar to those described by Anderson [2]

Nearly every arch removed has had its own peculiarities. The right and the left sides often are markedly dissimilar. The defects may be anything from linelike separation to total absence of the pars interarticularis Separate small-bone fragments frequently are found in the fibrous tissue between the facets Not infrequently the bony portions of the pars are elongated and lie in a direction that is away from the superior facet. Often such projections are directed vertically toward the nerve roots This would

indicate a process of very long standing which must have occurred no later than the active growth period of the bone.

The inferior facets often are asymmetric and lie in other than the accepted plane. Spina bifida of varying proportions has been encountered in six cases. A common finding is an osteophytic growth of spurs about the facets and about ligamentous attachments.

Examination of the removed arches has failed to yield any evidence, either grossly or microscopically, of bone healing. Microscopic sections have shown a gradual change from bone to cartilage followed by ligamentous tissue, with none of the usual features of healing, such as of callus formations or eburnation of bony surfaces (Figs. 1 & 2).

In our operative material we have not encountered any patients with multiple free arches as described by Roche and Rowe,[21] although we have seen a patient who had mild back pain with roentgenographic evidence of free arches at L-4 and L-5.

SYMPTOMATOLOGY

As mentioned above, the principal complaint of patients is low backache with or without sciatica. Often the pain may radiate into one or both lower quadrants or inguinal regions. (It is worthy of note that we have several patients who have had abdominal operations because of this distribution of pain, the back disorder having been overlooked.) Vague hip, buttock and leg pain is another frequent complaint.

Not infrequently the discomfort is very mild and only associated with heavy work involving lifting, bending, stooping and twisting. We have been unable to arrange our patients' complaints into any specific symptom complex.

Since almost the sole complaint of the patient is pain, we have speculated on its production. Almost certainly the back pain, soreness and tenderness would be due to irritation of the soft tissues resulting from the hypermobility of the arch. The stretching of attached ligaments of the mobile arch seems to be the most obvious explanation. Sciatica predicates pressure on one or more of the roots of the sciatic nerve. This is brought about in three ways, or a combination of them:

First, the superior portion of the free pars interarticularis may be forced down against the root, which lies only a few millimeters directly below it. Flexing the back bows the heavy paraspinous muscles which force the free piece ventrally. The same motion pulls the spinous process cephalad, which tips the pars interarticularis downward against the roots. These motions can be demonstrated easily by making a working model of the lower spine, using several vertebra separated by "intervertebral disks" of sponge rubber and rubber bands to simulate muscle and cutting the pars interarticularis of L-5 with a saw. Flexing the spine gently will demonstrate graphically how the free arch falls forward onto the underlying roots.

Second, a factor that may produce sciatica is a herniation of the intervertebral disk. In this disorder it need not protrude as far as in an intact spine to produce root pressure, as the bony clearance due to the free arch is less.

Third, a mechanism has been pointed out by Adkins.[1] Ridges of bone formed at the disk margins may constrict the nerve roots as they pass over the vertebra and through the root canals. Presumably these arise following trauma or "scarring" of the intervertebral disk.

The contention that the pain is related to vertebral body slippage appears to be untenable. First, very few cases have been reported of observed and measured slippage during the symptomatic phase. Second, often the most painful backs show no vertebral body displacement, only spondyloschisis. Third, at operation we have been unable to shift the body. Finally, asymptomatic displacement even to Grade 4 has been noted. Certainly the grade of displacement of the vertebral body and the patients' complaints have no correlation. Unfortunately,

many persons with spondyloschisis have been pronounced malingerers because the defect was not detected. Conversely, pre-employment studies are revealing great numbers of totally asymptomatic people who have done heavy manual labor for years and exhibit all degrees of slippage. Our own observations lead us to believe that patients should be treated on their symptoms alone, and not on the roentgenographic findings. We think that the preoccupation with the position of the vertebral body has been out of all proportion to its importance in the treatment of this disorder.

DIAGNOSIS

The diagnosis of spondyloschisis and Grades 1 and 2 of spondylolisthesis rests entirely on roentgenographic studies. In the more serious grades of the latter, the marked lordosis of the back, the increased prominence of the L-4 or the L-5 spinous process and pelvic examination in women may allow the diagnosis to be made, roentgenograms serving as confirmation.

Besides the conventional anteroposterior and lateral views, 45° oblique roentgenograms are a necessity to demonstrate the defects of spondyloschisis. Even other angles may be necessary at times. We have seen many cases that were missed because of the lack of these views, and now we are including oblique roentgenograms routinely in our study of every patient with a low back disorder. Reliance cannot be placed on the older methods of diagnosis such as Ullman's line, Bowman's bowline and posterior margin check.

We still find it necessary on occasion to explore a painful low back without a definite preoperative diagnosis. The loose arch is evident as soon as the lumbar fascia is incised. The first case of spondyloschisis that we encountered was such an exploration. It must be acknowledged that a "normal" roentgenographic study of the low back does not exclude the possibility of spondyloschisis.

TREATMENT

Many methods of treatment have been advocated, based principally on the individual doctor's conception of the cause of the symptoms, the progress of the disease, the patient's complaints and the innate ability of the body to heal itself.

For a long time it has been recognized that many patients require little treatment and that all do not progress to a Grade 4 spondylolisthesis with incapacitating pain and/or neurologic defects! Hence conservative measures such as back supports, exercises and change of jobs have been used. However, a large group of patients have remained unhappy from a symptomatic standpoint. Reporting on 50 cases that were treated conservatively, George found only 18 per cent "cured" and 62 per cent unimproved, while Caldwell, commenting on a small series (26 cases), felt that seven had "good" and twelve had only "fair" results. This is in line with our experience and indicates that the patient with symptomatic spondylolisthesis or spondyloschisis is dissatisfied more often than not with conservative measures.

Besides the patients' continued complaints of back and leg pain, another factor that has led to an operative approach to the disorder has been the fear of progressive slip of the vertebral bodies and anticipated neurologic damage. This fear has been voiced loudly for years, both in the written and the spoken word of many orthopaedists, but few actual data concerning the frequency, the age, the time and the degree of slippage ever have been published It is our opinion that spondylolisthesis is more common than spondyloschisis, that generally the slip occurs early in life, and that in itself it is almost painless and the vertebra almost always stabilizes itself. At operation we have never discovered a vertebral body that was not firmly fixed and almost immobile.

Because of failure to understand the exact nature and course of the disease, fusion of some sort now is the vogue when conserva-

tive measures fail. Assuming that the symptoms stem from an "unstable" vertebral body, it has long seemed logical to us that some kind of anterior fusion should be the most successful. There have been sporadic reports of such procedures, including the use of large strips of bone, staples and even bone pegs driven through the body of the sacrum into the fifth lumbar vertebra, but they never have found favor, principally due to the attendant technical difficulties encountered at operation. (Speed[22])

Some form of posterior fusion usually is advocated. These are easy to do, but solid bony union occurs in from only 50 per cent to 85 per cent, depending on which author one consults. Most physicians concur that this method of treatment leaves much to be desired. Meyerding,[19] in his series of 143 operations, reported only 60 per cent relieved completely. Briggs and Keats,[5] Caldwell[6] and many others have commented on the fact that often patients complain of back and leg pain despite roentgenographic and clinical evidence of solid bony union.

Colonna[8] recently reviewed a series of patients operated upon by himself. Of 29 patients who were fused, 20 reported excellent results (71.4%).

It was Dandy's[10] contention that the pain was due to root compression incident to a protruding intervertebral disk. He was one of the early advocates of disk removal without fusion and stated that it was not necessary in spondylolisthesis. Often removal of the disk alone was not the solution of the patient's complaints. However, others began to realize that root compression was an important factor. Briggs and Keats[5] advocated foramenotomy, hemilaminectomy or complete laminectomy of the loose lamina in those patients with leg pain. Many orthopaedists now urge complete removal of the free neural arch before proceeding with the fusion. Chandler[7] was one of the first to report this, although only as a matter of convenience at operation rather than as a principle to be followed.

Caldwell[6] must be credited with first pointing out a new approach to treatment. He urged that when sciatica was present the nerve roots be explored and decompressed by removal of a protruding disk and/or bone. In concluding his analysis he states:

> It seems possible, from a theoretic viewpoint, that in certain cases the symptoms might best be relieved by removal of the attached neural arch and the inferior articular processes. Liberation of the nerve roots without fusion of the articulations or bridging of the defect with a bone graft should, of course, be supplemented by postural exercises and low back supports.

Just which were the certain "cases" and why the need for back supports is not stated, but it indicates less apprehension about the vertebral body.

James and Nisbet[15] appear to have realized the importance of the free arch in the production of symptoms. They reported six cases of removal of the arch and associated protruding disk with an intervertebral fusion. Their results were uniformly good. What seems even more important, in their paper they present what we believe to be the first case reported in the literature of removal of the free posterior arch with complete relief of symptoms, no other treatment having been carried out.

Being aware that many patients with spondylolisthesis go through life with few or no symptoms and that increasing numbers of people with asymptomatic spondyloschisis are being uncovered in pre-employment roentgenographic examinations of backs, we came to the conclusion that people with these anomalies did not have "unstable backs" per se. In the course of exploring low backs for intractable pain and/or sciatica, we encountered several patients with free arches. We were struck by their excessive mobility, and it was easy to demonstrate that by pushing the spinous process cephalad, rotation occurred at the inferior facets and that the pars interarticularis attached to the arch impinged directly on the underlying nerve roots. This was especially evident if,

in addition, there were a protruding disk and/or a free piece of bone in the fibrous tissue between the arch and the superior facets. We felt certain that these structures must be the cause of the patients' complaints.

Once having grasped this concept, we removed the arch *in toto*, together with a protruding disk if present. The vertebral body then was in plain view. To our surprise we found that it was always fixed exceedingly firmly to the adjacent sacrum; little or no motion could be produced by trying to rock it or even by pounding on it with a hammer. Fusion in such a situation seemed superfluous; therefore, it was logical to dispense with it entirely. This shortened the operation and allowed us to mobilize our patients immediately, thus reducing possible postoperative complications. There also seemed to be no point in restricting their postoperative activities.

Our conclusions were strengthened by a review of nine patients on whom we operated in 1952. In each instance the free arch was removed and a combination fusion was carried out using chip grafts and cortical strips of bank bone. Solid fusion, as determined by means of lateral bending, flexion and extension roentgenograms, was demonstrated in only 33 per cent of patients. Of the six patients who did not have a solid fusion, five (83%) were relieved of all symptoms, and the remaining patient was markedly improved. This led us to the belief that the added stability (?) obtained by solid fusion did not add materially to the relief of symptoms.

We had removed several free arches before we were aware of the paper written by James and Nisbet,[15] nor were we aware that Gill[13] had reached the same conclusion before us and for some time had been carrying out the identical procedure with excellent results.

CRITERIA FOR OPERATION

Since the body stabilizes the "free" vertebra in the majority of people with bilateral defects in the pars interarticularis, no treatment is indicated in those who are asymptomatic; nor has it been proved that restriction of activities is helpful as a prophylactic measure.

Those patients who have only mild symptoms and are happy with back supports and the results of corrective exercises need nothing further. Any others who are tired of their back and leg complaints, have lost time from work and have been told honestly about the risks of surgery and the chances of relief are candidates for surgery. Age is no bar. Older people with sciatica suffer as much as youngsters. The risk and the time in bed are so small that it seems unfair to deny relief to anybody except those who are in the most precarious condition.

Our method of treatment is simple. The free posterior arch is dissected out in one piece. This is a simple surgical feat. The second step is to explore carefully the region between the facets, as often additional unattached pieces of bone are embedded in dense fibrous tissue overlying the roots. These may represent unfused (?) portions of the pars interarticularis. These should be searched for and excised. Next, the intervertebral disk is inspected. If it has ruptured, or if it is soft and fluctuant, it should be removed. This is accomplished as thoroughly as possible, taking not only the nucleus pulposus but also all the available cartilage and fibrous tissue (what has been removed cannot recur). Finally, the foramina are scrutinized, and any bony prominences that may impinge on the roots are ronguered away. This latter point has been brought out clearly by Adkins.[1]

When all skeptical assistants are convinced that the roots are free, the wound is closed in the same way as after an ordinary laminectomy. The operation rarely takes more than one hour, and the blood loss is small.

No special postoperative treatment is needed. The patient is ambulatory from 24 to 48 hours after operation. Braces, supports or casts are not required. Discharge

from the hospital usually is 7 to 10 days after operation. Restriction of activities is minimized. Full activities are allowed from 4 to 6 weeks after simple arch removal. Should an intervertebral disk be excised in addition, we advise against heavy work for 3 to 4 months after operation.

DETAILS OF OPERATION

Operation is done in the prone position, the legs flexed about 45°. Hypobaric spinal anesthesis is used. A mid-line incision about 6 inches long is centered on the bone which has the free arch. The lumbar fascia is incised close to the spinous processes of the adjacent as well as the affected vertebra. The muscles are stripped subperiosteally off the spinous processes, lamina and the pars interarticularis and facets. The free arch then is conspicuously mobile.

With a scalpel, the interspinous ligaments are severed, and the ligaments about the inferior facets are divided. The spinous process is grasped with a heavy clamp, and the arch is rocked forward. The ligamentum flavum is dissected off the arch with a sharp periosteal elevator. The arch then is rocked backward, and the superior edge of the arch is freed similarly of its ligament.

The arch next is rocked to one side, and the fibrous tissue is scraped off the pars interarticularis. There is a band of dense fibrous tissue at the site of the break in the pars interarticularis; this must be cut and later excised. Care must be taken to preserve from injury the nerve root that underlies this band. When this maneuver is carried out bilaterally, the arch can be lifted out in one piece. If care is exercised, very little bleeding occurs, and transfusions rarely are required.

When the field is dry, the dense fibrous tissue freed previously from the pars must be carefully excised up to the superior facet. Often it contains unattached fragments of bone which may be ununited portions of the pars interarticularis.

The intervertebral disks at L-4 and L-5 now can be inspected and removed if necessary. The foramina should be surveyed carefully, and, if any spicules of bone seem to impinge on the nerve roots, they should be excised. Curiosity about the mobility of the vertebral body can be satisfied by trying to move it. We have found that it cannot be budged. The incision then is closed in layers, as in any laminectomy.

RESULTS

The clinical results have been almost uniformly excellent. Backaches and sciatica are relieved. The back remains mobile. Men and women return quickly to their occupations, even manual laborers.

During the present follow-up period, we have not detected any further shift of the vertebral body of any patients. We recognize that this may occur, although we feel that the mechanics of the back have been unchanged by the removal of the free arch. It has been noted infrequently that further anterior slip of the vertebral body has occurred in untreated patients; therefore, we may assume that it can happen after removal of the free arch, but with no increased frequency. However, since the vertebral body always has been found to be firmly fixed at operation, any subsequent movement must be very slow.

We have not closed our minds to the possibility that fusion may be necessary in the future. Should an alarming slip occur, fusion might be in order, although the actual slip of the fifth lumbar over the edge of the sacrum is exceedingly rare, and, when it does, usually it produces no neurologic defects. However, should fusion seem to be indicated, we would consider an anterior approach.

The only other indication for fusion would be a recurrence of backaches and/or sciatica not due to root impingement by another protruding disk, or remnants of one removed previously, or remnants of a free-arch. We are inclined to think that one of these probably would be the cause of recurrent pain rather than an "unstable back."

During the 1951 to 1952 period we oper-

TABLE 1. SPONDYLOSCHISIS

Age at onset of symptoms	Number of cases	Sex	*Protruding disk* at operation	*Onset of* symptoms before operation	History of injury to back	Presence of sciatica
10-20	—	—	—	—	—	—
20-30	2	M 1 F 1	0	Both about 2 mos.	Yes 1 *No 1*	2 present
30-40	4	M 1 F 3	2	30+ yrs.; 2 yrs. 2— 1 yr.	Yes 1 No 3	2 present 2 absent
40-50	3	M 2 F 1	1	3 yrs., 6 mos 3 mos.	Yes 2 No 1	2 present 1 absent
50-60	1	M 1	0	Many years	Yes	present
60+	—	—	—	—	—	—
Total	10	*M 5* *F 5*	3		Yes 5 No 5	Yes 7 No 3

TABLE 2. SPONDYLOLISTHESIS

Age at onset of symptoms	Number of cases	Sex	Protruding disk at operation	Onset of symptoms before operation	History of *injury to* back	Presence *of* sciatica	Degree of slip
10-20	2	M 1 F 1	0	6 mos. 4 yrs.	0	0	1st 1 *3rd 1*
20-30	1	F 1	0	1 yr.	0	Yes	3rd 1
30-40	4	M 3 *F 1*	Yes 2 No 2	Many years, 2 yrs , *1½ yrs., 6 mos.*	*0*	*Yes 4*	*1st 4*
40-50	6	M 4 F 2	Yes 1 No 5	27 yrs., 3 yrs., 1 yr., remainder "years"	0	Yes 4 No 2	1st 5 2nd 1
50-60	3	M 2 F 1	0	"Years," 18 yrs., 1 yr.	Yes 2 No 1	Yes 2 No 1	1st 3
60-70	*3*	*M 3*	*0*	24 yrs., "years," 20 yrs.	Yes 2 No 1	Yes 2 No 1	*1st 3*
70+	—	—	—	—	—	—	—
Total	19	M 13 F 6	3		Yes 4 No 15	Yes 13 No 6	1st 16 2nd 1 3rd 2

ated upon nine patients with removal of the free arch followed by a modified Hibbs type of fusion as related above. Beginning with the year 1953, no patients have been fused, operations having been restricted to removal of the free arch and/or excision of an offending intervertebral disk, if present. Nine such operations were performed during that year All nine patients were re-examined and studied roentgenographically 2 years after surgery. All are at work, only one housewife reporting limitation of her activities. Two had diseased disks which were removed at the same time, one at L-5 to S-1 interspace and one at the L-4 to L-5 interspace. Only the housewife, with a lawsuit pending, has significant complaints These are: persistent backaches, recurrent episodes of right sci-

atica and transient episodes of numbness and prickling in the lateral calf of the right leg. Examination discloses limitation of flexion of the back and a positive straight-leg-raising test on the right at 60°. This discomfort is much less than before operation, and the patient does almost all her housework. Reoperation has been declined.

The remaining patients complain only of occasional mild backaches and some "stiffness" on sitting or on arising in the morning. Generally the symptoms are less than encountered in those patients who have undergone a lumbar laminectomy for other reasons. Follow-up roentgenographic studies failed to disclose any further shift of the vertebral bodies. Flexion and extension roentgenograms demonstrated only minimal motion of the L-5 vertebra in two patients.

During the years 1954 and 1955, 19 additional patients were operated upon. Only two patients have had any recurrence of symptoms. A man complains of a moderate amount of backache and a mild unilateral sciatica following a day of fairly heavy manual work. These symptoms appeared 9 months after operation. Physical findings have not been impressive, and there are no further roentgenographic changes. This patient may require additional therapy in the future.

A second patient returned 3 months after operation with the complaint of acute onset of pain and numbness on the lateral side of the right lower leg, together with moderately severe backaches. These appeared after doing some light work. During the course of the next 6 months the discomfort persisted, numbness in the area of the fifth lumbar dermatome appeared, and atrophy of the anterior tibial muscle was noted. Conservative measures were of no avail. The back was re-explored, and to our chagrin we found a bony fragment about 7 mm. in diameter and from 2 to 3 mm. in thickness lying directly over the L-5 root. Repeated roentgenograms had failed to disclose it. Simple removal was carried out. Inspection of the disks failed to disclose any protrusions. The patient has been free of pain since the second operation, but the numbness and the atrophy persist.

When we first began to remove the posterior free arch and decompress the adjacent nerve roots, we were not cognizant of the work of Gill.[13] Recently his excellent paper has been published. It is gratifying to learn that his experiences are identical with ours. The longer follow-up period tends to confirm the earlier impression that nearly all patients are relieved of all major symptoms, return to their occupation and show no further bony changes.

We have not resorted to his technic of manipulating backs in the postoperative periods. Neither have we been impressed by the necessity of removing additional spinous processes.

In summary, of the 29 patients who have had only the free arch removed, one patient has had severe enough symptoms to warrant reoperation, at which time we found bony fragment overlying the L-5 nerve root that had been overlooked. One patient has many complaints but very minimal findings and has refused further treatment. A third patient, who is a manual laborer, has had mild backaches and sciatica but was working without loss of time when seen last.

REFERENCES

1. Adkins, E. W. D.: Spondylolisthesis, J. Bone & Joint Surg. 37:48-62, 1955.
2. Anderson, K. J., and Breck, L. W.: Spondylolisthesis; rare variant with unilateral isthmus defect and slight displacement, South. Surgeon 15:470-477, 1949.
3. Baker, D. R., and McHolick, W. J.: Spondylolysis and spondylolisthesis in children. In preparation.
4. Batts, M.: The etiology of spondylolisthesis, J. Bone & Joint Surg. 21:879-884, 1939.
5. Briggs, H., and Keats, S.: Laminectomy and foraminotomy with chip fusion; operative treatment for relief of low-back pain associated with spondylolisthesis, J. Bone & Joint Surg. 29:328-334, 1947.

6. Caldwell, G. A.: Spondylolisthesis: analysis of 59 consecutive cases, Ann. Surg. 119: 485-497, 1944.
7. Chandler, F. A.: Lesions of the "isthmus" (pars interarticularis) of the lamina of the lower lumbar vertebra and their relation to spondylolisthesis, Surg., Gynec. & Obst. 53:273-306, 1931.
8. Colonna, P. C.: Spondylolisthesis, J.A.M.A. 154:398-402, 1954.
9. Congdon, R. T.: Spondylolisthesis and vertebral anomalies in skeleton of American aborigines with clinical notes on spondylolisthesis, J. Bone & Joint Surg. 14:511-524, 1932.
10. Dandy, W. E.: Treatment of spondylolisthesis, J.A.M.A. 127:137-139, 1945.
11. Friberg, S.: Studies on spondylolisthesis, Acta chir. scandinav. 82 (suppl. 55): 1, 1939.
12. George, E. M.: Spondylolisthesis, Surg., Gynec. & Obst. 68:774-781, 1939.
13. Gill, G. G., Manning, J. G., and White, H. L.: Surgical treatment of spondylolisthesis without spine fusion, J. Bone & Joint Surg. 37:493-520, 1955.
14. Hitchcock, H. H.: Spondylolisthesis: observation on its development, progression and genesis, J. Bone & Joint Surg. 22:1-16, 1940.
15. James, A., and Nisbet, N. W.: Posterior intervertebral fusion of the lumbar spine, J. Bone & Joint Surg. 35:181-187, 1953.
16. Kilian, H. F.: De spondylolisthesi gravissimae caussa. nuper detecta commentatio anatomica-obstetricia, Bonn, George, 1854.
17. Kleinberg, S.: Spondylolisthesis and prespondylolisthesis, Arch. Surg. 27:565-587, 1933.
18. ———: Spondylolisthesis in an infant, J. Bone & Joint Surg. 16:441-444, 1934.
19. Meyerding, H. W.: Spondylolisthesis, Proc. Staff Meet. Mayo Clin. 9:666-671, 1934.
20. Neugebauer. Quoted by Speed.
21. Roche, M. B., and Rowe, G. G.: The incidence of separate neural arch and coincident bone variation, J. Bone & Joint Surg. 34:491-493, 1952.
22. Speed, K.: Spondylolisthesis; treatment (anterior bone graft), Arch. Surg 37:175-189, 1938.
23. Whitman. Quoted by Kleinberg. Vide supra.

Un Nove Tactica in le Tractamento de Spondylolisthesis e Spondyloschisis

Summario in Interlingua

Le autores discute in detalio le terminologia de spondylolisthesis e de spondyloschisis, lor origine (que illes reguarda como de natura congenite), lor pathologia, e le studios roentgenographic que es requirite pro establir lor diagnose. Le tractamento de iste condition consiste in le simple ablation del libere arco neural e del associate pecias de osso libere que jace possibilemente in le canal spinal. Esseva incontrate plure patientes con discos protrudente Istos es abferite simultaneemente, si necessari.

Vinti-nove patientes de iste genere ha essite operate. Le serie includeva solmente un non-successo, e isto resultava del facto que un libere fragmento de osso escappava al attention del operator. Duo altere patientes ha retenite minor symptomas de dorsalgia, sed illes non considera chirurgia additional como indicate.

Nulle nove dislocation del corpores vertebral esseva notate in ulle del patientes disponibile pro observationes posterior. Patientes in qui le arco libere es abferite pote retornar a lor travalio intra quatro septimanas. Patientes in qui un disco es abferite require un periodo de reconvalescentia alique plus extense. Nulle apparatos de supporto es requirite. Fusion spinal non esseva considerate como necessari o desirabile in ulle del casos.

21

Disability Evaluation in Degenerative Arthritis

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Accurate, equitable disability evaluation in arthritics who sustain a compensable injury is of the utmost importance to both physician and lay person. The problems of disability evaluation in persons who suffer from arthritis cannot be answered easily. Their solution should be decided by those best informed and in contact with such problems. Even then, with the present knowledge of the causation and our inability to predict infallibly the course of arthritis, occasionally an improper financial settlement will be made.

Disabling arthritis can be an economic tragedy and place a great burden on the victim. In present-day society there is an ever-increasing tendency to shift the financial phase of this burden to those persons, or group of persons, presently most able to pay. A large portion of this cost thus becomes the responsibility of the insurance companies.

The great social and economic importance of disabling arthritis is well known, and in recent years arthritic diseases have afflicted more persons than the total of those suffering from heart disease, cancer, tuberculosis and diabetes.[1,2,4,8] Approximately 97,000,000 work days annually are lost by self-dependent arthritic persons. Therefore, it is appropriate to develop a common system for evaluating industrial injuries in arthritics.

This chapter deals primarily with degenerative arthritis, although in many respects the comments are pertinent to rheumatoid and gouty arthritis. Degenerative arthritis presents the pathophysiology of articular cartilage fibrillar degeneration which is followed by incongruity of articulating surfaces. This incongruity is followed by stress changes in the articular capsule, leading to fibrosis and concurrently producing hypertrophic osteophytic changes at the synovial articular junction. Such degenerative arthritic changes apparently have their origin in traumatic insults in the joint, whether a single disrupting trauma or many small microtraumata to the joint surface or capsule. In their constitutional make-up certain people have a propensity toward developing degenerative arthritis, probably related to hereditary or metabolic states and not as a direct consequence of trauma, as previously defined.

There has been conflict in medical thinking regarding the traumatic relationship in arthritic disabilities. Positive evidence relating trauma to joint or adjacent soft-tissue changes is readily available in medical literature.[2,5,6,11] However, current literature reports virtually nothing to aid the practitioner in evaluating the degree or the length of disability in a given case of arthritis. The concept of disability in degenerative arthritis must be based firmly on pathologic physiology in this disease. The answer to these questions must come from specifically trained practicing physicians who encounter numbers of such problems daily. Many patients with arthritis claim etiology but actually have acceleration of the pre-existing disease, which may not have been symptomatic

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prior to the injury. It is not implied that persons who suffer a fracture with disruption of the joint surface fall into this category.

In one review of 500 industrial accidents 80 per cent of the patients were considered to manifest degenerative arthritis, and one half of these claimed industrial injury as the cause of disability.[3] Workmen's Compensation Commissions over the country currently hold that injury with resulting arthritis is fully compensable. At times the Commission has seen fit to allow the injured person to share that part of the responsibility for his disability that was inherent in his constitutional make-up.[9,10]

Two hundred District Court opinions which were appealed and in which medical testimony as to the origin of and resulting disability from arthritis was a deciding question were reviewed by the authors.[7] Those physicians skilled in the management of arthritis testified regularly that in the case under consideration there was a propensity toward degenerative arthritis and that the injury was an aggravating, or accelerating, factor. Of necessity, judicial decisions consider all facts in cases at hand and regard medical opinion as a modifying factor but not as unalterable dicta; thus, conflict of legal opinion and scientifically considered medical opinion will result at times.

A questionnaire was submitted to 197 practicing physicians, both rheumatologists and orthopaedic surgeons, who in their daily practice encounter a large number of medicolegal problems The purpose of this poll was to discover the current medical thinking on these matters. The questions were worded so as to ascertain how the practitioner evaluated specific problems in which a patient alleged traumatic origin of traumatic illness. Of 197 questionnaires, 80 were mailed to orthopaedic surgeons and 117 to rheumatologists. The physicians selected in each group were those who had by conversation, by personal contact, previous publication or specific type of practice expressed an interest in this facet of arthritic study. Forty-eight orthopaedic surgeons and 96 rheumatologists responded.

SUMMARY OF THE QUESTIONNAIRES SUBMITTED

A. B. is a 60-year-old white male in excellent general health. He sustained a severe jarring injury to his neck while pursuing his regular work. The injury was not of the "whiplashing" nature. The patient's injury was covered automatically by Workmen's Compensation Statutes. He received excellent immediate first-aid and medical care, consisting of cervical traction, bed rest, physical therapy, etc. Roentgenograms 3 days after the injury showed advanced changes consistent with degenerative cervical arthritis.

The patient and his family deny categorically any history attributable to cervical arthritis prior to injury. Now, 1½ years after injury, the patient is entirely unable to do his usual work due to disabling symptoms of cervical degenerative arthritis. There is no reason to believe that the patient is malingering. All curative therapeutic approaches have been exhausted.

Question 1

Is it possible, probable or improbable that the degenerative arthritis was rendered symptomatic by the injury?

Eighty-two per cent of the rheumatologists and 88 per cent of the orthopaedic surgeons stated that they felt it was probable rather than possible or improbable that a person with previously demonstrable degenerative changes, yet without symptoms, could be rendered symptomatic by a single acute trauma.

Question 2

Do you consider symptomatic degenerative arthritis, alleged to be contributable to an injury, compensable to a previously asymptomatic individual?

Seventy-four per cent of the orthopaedic surgeons felt that this situation would be compensable, and 63 per cent of the rheumatologists agreed. Many comments suggested that token payments might be more proper, in that probably in the future the person would have had symptomatic degenerative arthritis.

QUESTION 3

Should a presently symptomatic arthritic who sustains injury as an aggravating influence be entitled to compensation for all subsequent disability pertaining to the arthritis?

Eighty per cent of the orthopaedic surgeons and 67 per cent of the rheumatologists stated that it would work an undue hardship on the liable person if such an attitude of allowing all future disability were to become a universal practice. Many commented that as regards employability, a major stigma would attach to susceptible individuals if compensation for subsequent disability were allowed. It should be noted that a significant number of physicians—one third of the rheumatologists and one fifth of the orthopaedic surgeons—did not feel that all subsequent disability should be attributable to the injury and, therefore, compensable as a direct result of the single trauma.

QUESTION 4

If all future disability is not compensable, should the responsibility for all disability be prorated between the persons liable for the injury and the patient?

Eighty-eight per cent of the rheumatologists and 86 per cent of the orthopaedic surgeons stated that some technic for prorating disability expenses between the injured person and the party responsible for that injury should be established. Several stated that it should be on an arbitrary basis, such as 50-50 or 25-75 per cent award. Most of them indicated that they would use some technic of weighing the evidence, both as to the pre-existing state of the disease and the extent of culpability of the injury, circumstance, etc. An attempt to ascertain how these physicians would assess and apportion the liability was requested, and they were asked to state their technic. Very frequently this question was left unanswered. Most of the responding physicians indicated that they would use a technic of weighing the factors of the disease severity, probably antecedent presence of degenerative arthritis, what the probable future course of the arthritis would be in their experience, the social status of the person, and the blameworthiness of the injured person in producing his own trauma.

DISCUSSION

In organizing the opinions of those polled and trying to relate this to information gleaned from the medical literature, it is apparent that those persons qualified to have an opinion would welcome a technic of prorating the social responsibility of traumatic degenerative arthritis in any one given case. It is apparent that many physicians use technics of this nature.

The concept postulated here considers that a patient at the time of injury had within himself "arthritis" at a set level of activity. This level might very well be an asymptomatic or a mildly symptomatic state. When trauma was superimposed, the arthritis already present moved to another level, this level being more symptomatic than the previous one, or, if the previous one was asymptomatic, it might now be symptomatic. This concept regards degenerative arthritis as a chronic progressive disease which, one day in the unknown future, will arrive at the level of symptomatology and/or activity of which the injured patient presently complains. When injury supervenes, the course of the arthritis may be modified. The injury is considered to be an accelerating influence upon an already present disease entity. Once the acceleration is interposed, the progressive disease moves to a new level of disability, which may remain static, as it might do when it arrived at this state in the unknown future.

The evaluation of this disability from a trauma then limits itself to an evaluation of the degree of acceleration of the entity already present. Of necessity the accelerating influence is intangible, but, when determined on the basis of sound consideration of established pathophysiologic aspects of the arthritides, an opinion can be derived satisfactorily. Generally it has been conceded that on the average degenerative ar-

thritis progresses at a similar rate. It must also be remembered that a large percentage of arthritics experience a cyclic pattern in the activity of their disease. This complicates even more the evaluation of their disability and requires a projected evaluation. The exact time in weeks, or months, that a given trauma will accelerate a given case of arthritis is a matter of speculation. However, it has been our experience that most cases of arthritis in which trauma has been superimposed reached a leveling-off period in from 6 to 18 months, depending upon the severity of the trauma.

It is considered that a person who develops degenerative arthritis usually has a constitutional defect within his physiologic system that permits him to develop arthritis. Virtually all persons manifest this to a certain degree. It is felt that ultimately he would have become a symptomatic arthritic. Concepts of this nature make the accelerating trauma an incident in the physiologic status of the individual sufficient to make his previous asymptomatic status now a symptomatic condition.

Basic and definite crystallization of medical thinking as to cause and effect of trauma in certain rheumatic diseases has been sustained. The poll of 197 rheumatologists and orthopaedic surgeons establishes a definite trend in thinking and affirms the thesis of this discussion. It is submitted that the most equitable way of evaluating an arthritic who is rendered symptomatic, or whose symptoms are accelerated by an injury, is a technic to establish the patient's present symptomatic and clinical state and at what future date this particular patient would most likely arrive at his present symptomatic and clinical state had he had no injury. The difference between these two times is the accelerating factor of the injury that he sustained. This, then, would be the suggested time of temporary total disability for which the injurying party should be liable. This accelerated time usually is from 6 to 18 months, but it is emphasized that every patient must be considered individually, and all facets of the patient's problems must be weighed carefully.

REFERENCES

1. Bennett, G. A., Wayne, E., and Bauer, W.: Changes in the Joint at Various Ages with Particular Reference to the Nature and Development of Degenerative Joint Disease, New York, Commonwealth Fund Report, 1952.
2. Hanlon, D.: Osteoarthritis aggravated by trauma, Am. J. Surg. 78:556, 1949.
3. Hench, P. S.: Rheumatism review of American and English literature, Am. J. Med. 28:66, 1948.
4. Hollander, J. L., *et al.*: Arthritis and Allied Conditions, ed. 5, Philadelphia, Lea & Febiger, 1953.
5. Morehead, John: Activation of disease by trauma, New York J Med. 38:20, 1938.
6. [illegible] of Trauma, ed. 1,
7. [illegible] tions and Summary of all State Supreme Court Decisions. New York and San Francisco, West Publishing Co
8. Preliminary Reports of National Health Survey: Sickness and Medical Care Series Bulletin No 6, Washington, D. C., U. S Public Health Service
9. Proceedings—37th Annual Convention International Association of Industrial Accident Board and Commissions: U S Department of Labor Bulletin No. 156, 1954
10. Snapper, I. M · Personal communication.
11. Wolfe, E. Traumatic arthritis, Indust. Med. 17:40, 1948.

Evalutation del Invaliditate in Arthritis Degenerative

Summario in Interlingua

Iste articulo summarisa le resultatos de un questionario distribuite a un grande numero de orthopedistas e rheumatologos con le objectivo de colliger informationes in re lor ideas concernente le evalutation del invaliditate in arthritis degenerative. Le questiones

submittite a illes concerneva lor conception del causation de arthritis degenerative, del relation inter arthritis degenerative e trauma, e del technicas pro le evalutation del effecto de trauma in le production specific de invaliditate. Finalmente, le questionario habeva le function de probar le opiniones del questionatos in re le duration probabile del invaliditate e in re le question de qui es responsabile pro pagamentos de invaliditate quando un tal es presente.

Super le base del responsas recipite e del lectura de pertinente publicationes medical e forensic il pare necessari acceptar arthritis degenerative como un entitate de morbo inherente. Es stipulate que le trauma superimponite es un factor accelerante in le historia natural del morbo e non un factor aggravante. Per consequente le proposition es formulate que le invaliditate es determinate secundo le grado de acceleration e non secundo le grado del invaliditate total. Un tal plano es eque pro omne personas concernite con le invaliditate.

22

Posterior Elementectomy in Ankylosing Arthritis of the Spine*

CLYDE W. DAWSON, M.D., F.A.C.S.†

In 1945 Smith-Petersen, Larson and Aufranc[5] first reported the use of osteotomies of the lumbar spine as a surgical treatment for the flexion deformity present in rheumatoid spondylitis. Their method employed the removal of small wedges of bone by means of an osteotome from across the ankylosed facets, beginning along the margins of the lamina. Later, these wedges were used as bone grafts on a prepared bed over the lamina. In 1947 Briggs, Keats and Schlesinger[2] reported on the use of the wedge osteotomy of the lumbar spine for correcting this deformity. In 1946 La Chapelle[3] reported a two-stage osteotomy of the spine for deforming rheumatoid ankylosis. This method combined a posterior wedge osteotomy across the lamina, the facets and the spinous processes, followed by an osteotomy of the body of the vertebra with the insertion of a bone graft between the opened segments of the body. In 1952 Adams[1] again described in detail the wedge osteotomy and stated that the procedure was best carried out with the patient lying on his side. He employed a special apparatus to extend the patient following the osteotomy. This had the general appearance of an osteoclasis apparatus.

The indications, the dangers and the safeguards, as well as the judgment and the skill necessary in the surgical treatment of ankylosing spondylitis, were reported carefully by Adams in 1952. With wider experience in the care of the arthritic patient and the improvement in medical control of the active stages of the disease, I find it expedient and more desirable to carry out certain surgical procedures at an earlier stage of the disease than formerly was thought to be possible.

In considering the surgical correction of an ankylosing deformity of the spine, it is important to realize that the duration of the deformity, as well as the location of the maximum flexion deformity, is a governing factor in the success of any surgery. In most instances in ankylosis of long standing, the soft parts, the ligaments and the muscular attachments are exceedingly fibrotic, if not completely ossified, so that identification of structures is very difficult, if not impossible. When ossification of the anterior vertebral ligament has occurred or has been completed, frequently the success of a single-stage procedure is jeopardized. Then it becomes necessary to employ the multiple-level or multiple-stage osteotomy. The lumbar area is the site of election for correction of the deformity whenever possible. The absence of the rib cage makes correction much easier, and, with termination of the cord at the 1st lumbar level, trauma to nerve tissue is less likely below this level. The flexion deformity in ankylosing spondylitis is usually most severe in the lumbar area. There is the loss of the normal lumbar lordotic curve, and often there is a complete absence of the sacral angle. The exact level

* Read before the Russell Hibbs Society by Dr. Judson D. Wilson on May 4, 1956.

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chosen for correction between the 1st lumbar and the lumbosacral junction depends upon the angle of the sacrum and the presence of any complicating scoliosis. In the author's experience, the 4th lumbar level lends itself to greater correction of deformity and the restitution of the more normal appearing trunk and extremities postoperatively than can be obtained at higher levels.

When one approaches the ankylosed spine surgically, it is very important to position the patient properly on the operating table. I personally prefer the use of the fully prone position. This requires that intratracheal anesthesia be used. The table must be placed in maximum flexion, and the patient must be supported by rolls under the shoulders and across under the pelvis to give adequate support and a good airway without strain on the dorsal or cervical spine. I would not attempt such surgery without the skilled assistance of a completely competent anesthesiologist.

A mid-line incision is employed. The spinous processes and the supraspinal ligaments are visualized from the 2nd sacral to the 2nd lumbar level. This wide exposure simplifies identification of landmarks, even those obscured by ossification of ligamentous structures. The spinous process of L-4 is removed by ronguer, and the interlaminar area between the 4th and the 5th lumbar is identified. The lamina of the 4th lumbar is completely removed by means of a ronguer and the use of a Kerrison punch. A dural elevator is employed to separate the dura from the ossified ligamentum flavum and lamina. Extreme care must be employed in these steps to protect the dura and the nerve root from either pressure or trauma. The foramen at the 4th to the 5th lumbar level is identified and located. The facets, both the inferior of the 4th lumbar and the superior of the 5th lumbar, then are excised completely, the intervertebral foramen being exposed bilaterally. As much of the pedicle of the 4th lumbar is removed as becomes necessary to obtain the correction desired. As the bony lamina, the facets and the pedicular margins are removed, it is interesting to note that the patient's deformity begins to correct

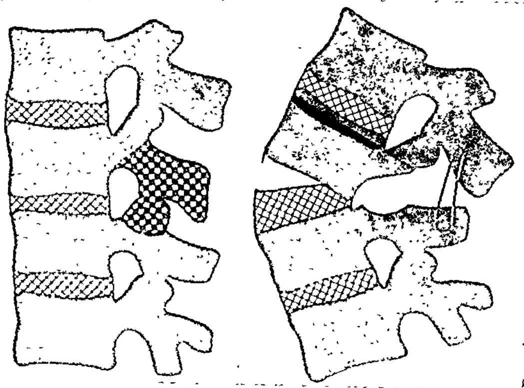
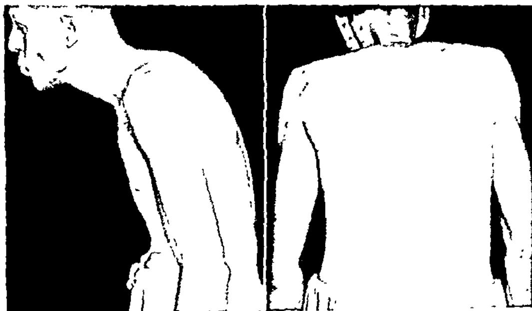


FIG. 1. Schematic drawings *(left)* of posterior elementectomy and *(right)* of manner of fixation posteriorly.

spontaneously (Fig. 1). This continues until the patient is resting firmly on the operating table, even though originally it was thought that he had been well supported with no evidence of stress at either extremity of his flexion deformity. Once the determined amount of lamina and pedicle is removed, the table is leveled flat gradually, thus allowing the deformity to continue to correct under direct vision. In the author's experi-



FIGS. 2 to 5, Case 1. Fig. 2 *(Above)*. Side and back views preoperatively.

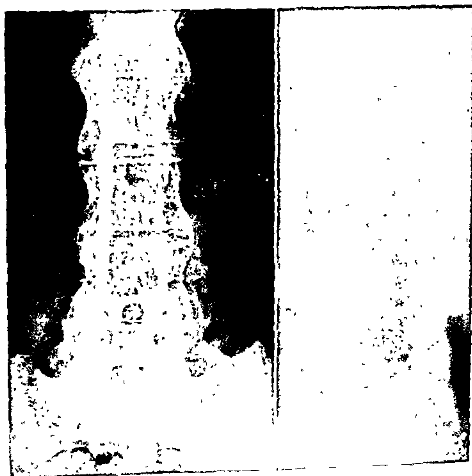


FIG. 3. *(Left)* Anteroposterior and *(right)* lateral roentgenograms taken preoperatively.

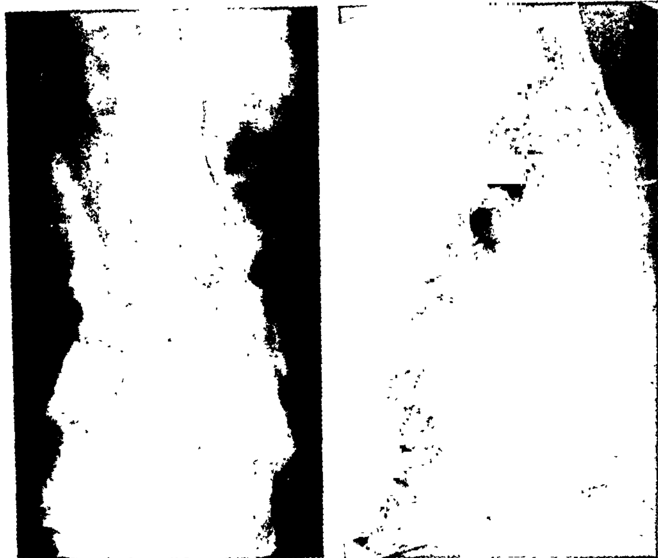


FIG. 4. Lateral roentgenograms taken *(left)* 2 weeks and *(right)* 6 months post-operatively.

ence this occurs without effort or undue incident at the 4th lumbar level until the spinous process of the 3rd lumbar is in close proximity to the spinous process of the 5th lumbar. This may not seem to be logical until one considers that there still remains a severe flexion deformity at the 5th lumbar to the 1st sacral level.

Stability of the spine laterally is excellent at the completion of this phase of the surgery. Prevention of a recurrence of the deformity is required. I have maintained the position of correction by wiring the spinous process of the 3rd lumbar to the spinous process of the 5th lumbar. This wire is inserted through the base of the 3rd lumbar and near the tip of the 5th lumbar, thus producing a lever effect that prevents any forward displacement of the 3rd and the 4th lumbar on the 5th lumbar. This method first was described by Rogers[4] in 1942 to preserve the reduction of a dislocated cervical vertebra.

The term *posterior elementectomy* is used here in preference to the term *osteotomy* or *wedge osteotomy* because I believe that it describes more accurately what is done. The above description makes it readily understood. No effort is required to correct the deformity in the region of the vertebral body. With the spontaneous correction following the posterior elementectomy, there is a wedging open that occurs between the level of the inferior end-plate of the vertebral body and the intervertebral disk. No posterior displacement occurs because the pos-

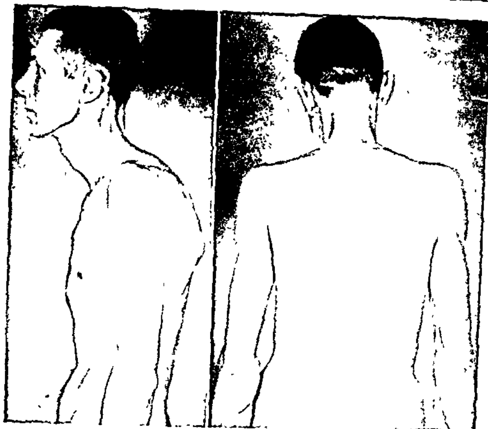
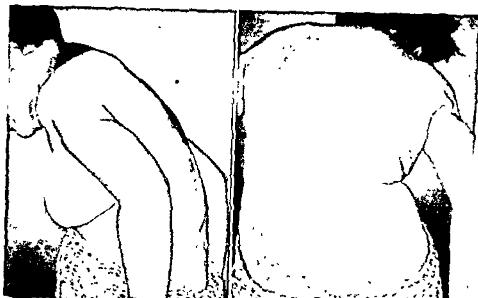


FIG. 5. Side and back views taken 3½ years postoperatively.



FIGS 6 to 9, Case 2. Fig 6 (*Left*). Side and back views taken preoperatively.

terior vertebral ligament is intact. There is no need to place a bone graft in the anterior defect so produced because it fills in rapidly with new bone.

In ankylosing spondylitis, ossification is accelerated. I have not resorted to the use of any bone-grafting material across the surgically produced posterior defect because it is my opinion that the disease process itself will accomplish a rapid reossification. The follow-up roentgenograms demonstrate adequately the reason for this opinion.

The summary of two cases demonstrating the procedure described above are presented for descriptive purposes. No attempt is being made in the short space of this chapter to analyze a series of end-results.

Case 1 (H. H.), first seen at the age of 33 years, a white male, chief complaint, inability

to stand erect. His onset of arthritis was 9 years prior to his first visit. Figure 2 shows his condition prior to surgery. Figure 3 demonstrates the amount of lumbar flattening and the angle of the sacrum prior to surgery. After being investigated thoroughly to determine the degree of activity of his arthritic process, which involved his spine only, he was taken to surgery on July 14, 1953. He underwent the procedure described above. His postoperative course was uneventful. He was kept in plaster shells for 8 weeks postoperatively, after which a Taylor-type back brace was applied, and he wore it for approximately a year. Since that date he has worn the brace only a part of the time. He was seen last in February of 1956, and at that time he stated that discomfort was limited to his neck because of stiffness in the region of the neck. Occasionally he had some aching in his back elsewhere. He still continues on a medical regimen for general supervision of his arthritic process. Figures 4 and 5 demonstrate the improvement in posture and the extent of ankylosis spontaneously.

Case 2 (G. O.), a 35-year-old white female who has been suffering from a rheumatoid arthritis with ankylosis of the spine. As will be noted from the illustrations, this patient has not only a flexion deformity but also a scoliosis complicating any corrective procedure. Her arthritic process has been under control for the last 2 years by the use of small doses of Cortone. It was felt that two operative procedures would be required to correct her deformity: the first to improve her flexion deformity and the second to improve her lateral curvature. Some correction of the lateral curvature immediately following the posterior elementectomy was anticipated.

The patient was admitted to the hospital, and a posterior elementectomy was carried out at the level of L-3, and the spinous processes of L-2 and L-4 were transfixed by wire suture at the completion of the operative procedure on October 17, 1955. Figures 6 and 7 show the condition of the patient preoperatively; Figures 8 and 9, postoperatively.

The patient's postoperative course was complicated by reactivation of the arthritic process in her left hip joint. It was found that even with a turnbuckle cast, in an attempt to correct the lateral curvature through the osteotomy site, no change in position occurred. The only correction was that accomplished at the time of

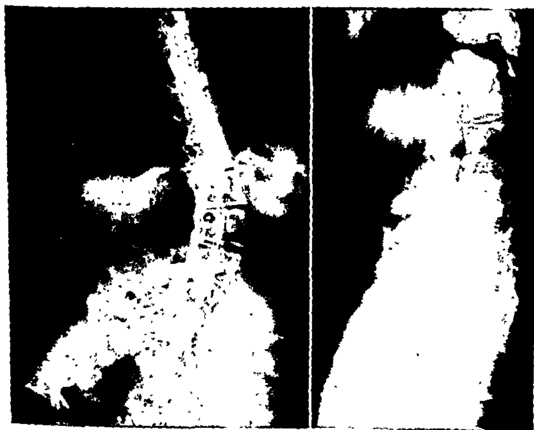


FIG. 7. *(Left)* Anteroposterior and *(right)* lateral roentgenograms taken preoperatively.

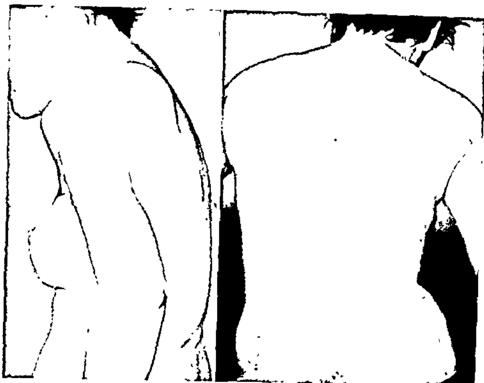


FIG. 8. Side and back views taken 1 year postoperatively.



FIG. 9 *(Left)* Anteroposterior and *(right)* lateral roentgenograms taken 15 months postoperatively

surgery with reference to the flexion deformity. She has had a much more stormy postoperative convalescence than *the first patient presented,* because her arthritis was not limited to the spine and because of the attempt to correct the lateral curvature. To date the patient is very pleased with the marked improvement in her flexion deformity alone and does not desire to have the

feels that she

In summary, (1) surgical correction of ankylosing arthritis of the spine is the procedure of choice in selected cases; (2) a

brief history of progress of such surgery is described; (3) the method employed in the cases above is described in detail, and it is felt that the term *posterior elementectomy* describes more accurately the technic employed.

REFERENCES

1. Adams, J. C.: Technique, dangers, and safeguards in osteotomy of the spine, J. Bone & Joint Surg. 34-B:226-232, 1952.
2. Briggs, H., Keats, S., and Schlesinger, Phillip T.: Wedge osteotomy of the spine with bilateral intervertebral foraminotomy, J. Bone & Joint Surg. 29:1075-1082, 1947.
3. La Chapelle, E. H.: Osteotomy of the lumbar spine for correction of kyphosis in a case of ankylosing spondylarthritis, J. Bone & Joint Surg. 28:851-857, 1946.
4. Rogers, William A.: Treatment of fracture dislocation of the cervical spine, J. Bone & Joint Surg. 24:245, 258, 1942.
5. Smith-Petersen, M. N., Larson, C. B., and Aufranc, O. E.: Osteotomy of the spine for correction of flexion deformity in rheumatoid arthritis, J. Bone & Joint Surg. 27:1, 1945.

Elementectomia Posterior in Arthritis Ankylosante del Columna Vertebral

Summario in Interlingua

In 1945, Smith-Peterson, Larson, e Aufranc[5] publicava le prime reporto del uso de osteotomias del columna vertebral como mesura chirurgic in le tractamento de deformitate flexional in casos de arthritis rheumatoide. In 1947, Briggs, Keats, e Schlesinger[2] reportava le uso del osteotomia cuneal in un forma plus o minus modificate, e La Chapelle[3] dava in 1946 un relation de un osteotomia biphasic del columna vertebral in casos de ankylosis rheumatoide. Un articulo per Adams,[1] publicate in 1952, describeva le methodo del osteotomia de cuneos.

Es presentate considerationes in re le indicationes, le periculos, e le mesuras de securitate in le tractamento chirurgic de un ankylosate columna vertebral. Es discutite le selection del sito pro le correction del deformitate e le rationes que justifica ille selection. Le methodo chirurgic usate per me como procedimento de election in osteotomia del columna vertebral ha essite le elementectomia posterior. Isto consiste in le ablation del seligite lamina, processo spinal, faciettas, e un portion del pedunculo del vertebra in que le correction del deformitate es attaccate. In le majoritate del casos, isto es les quarte vertebra lumbar. Post le ablation del desirate quantitate de lamina, facietta, e pedunculo, le correction es effectuate per un extension gradual del columna vertebral super le tabula de operation. Fixation es effectuate in le forma de un fixation a filo metallic inter le processos spinal del tertie e del quinte vertebra lumbar secundo le technica recommendate per Rogers[4] in 1942 pro le stabilisation de un vertebra dislocate.

Le termino *elementectomia posterior* es preferite proque illo describe plus adequatemente le manovras executate. Nulle effortio es facite a effectuar le fusion del vertebras interessate, proque tal fusion occurre spontaneemente post le tempore del intervention chirurgic, e etiam le defecto create inter le corpore vertebral e le disco se replena spontaneemente. Es presentate duo casos pro demonstrar in detalio le manovras technic usate e le resultatos obtenite.

Caso no. 1, un masculo blanc de 33 annos de etate, ha passate un periodo postoperatori de tres annos. Caso no. 2, un feminina blanc de 35 annos de etate, ha passate circa un anno depost le operation. Ambe iste patientes es marcatemente meliorate in lor postura. In ambe casos, le melioration postural ha meliorate le capacitate del individuo de persequer su eligite type de labor. Le beneficios obtenite ha meliorate le condition physic general del patiente e justifica le severitate del tractamento.

23

Intertrochanteric Fractures*

OTHO C. HUDSON, M.D., AND RICHARD P. GILIBERTY, M.D.

The problem in treatment of intertrochanteric fractures is not one of obtaining bony union, as is the case with intracapsular fractures, but a matter of preventing a residual varus deformity at the fracture site. The majority of these fractures unite. The residual impairment of function can be anticipated by measuring the amount of varus deformity. In general, it can be stated that the greater the varus deformity the greater the degree of functional impairment.

The ideal approach to the treatment of this fracture would encompass a method that would shorten the period of hospitalization and promote early ambulation of the patient. The fixation of the fragments by open reduction with the use of a mechanical appliance will fulfill all these requirements. To avoid the errors that may lead to failure of internal fixation, consideration has to be given to the mechanics involved.

In a nondisplaced linear type intertrochanteric fracture, the cortical surface area of opposition of the proximal and the distal fragments is adequate, so that the use of an angle plate will provide positive fixation. Rotation at the fracture site is prevented by the grip of the flange on the trabecular structure of the head and the neck. The normal angle is maintained because the vector of force of the muscle pull is directed along the same plane as the nail and in so doing compresses the opposing cortical surfaces at the fracture site.

When the fracture is comminuted to the extent that less than one third of the cortical surfaces of the two fragments remains intact, fixation with a flanged nail will prove to be more difficult. If the normal angle is restored by the purchase of the flange in the head and the neck and by the plate on the shaft, the comminution at the fracture site will result in the gapping of fragments. The constant muscle pull will exert enough force to close the gap and in so doing will cause the fracture to assume a varus position. Because the flange cannot be extruded, it will penetrate through the hip joint, so-called "overdrive,"[5] or actually bend or break the metal as illustrated in Figures 2 and 4.

In order to ascertain which of these fractures should be opened and fixed, they should be classified as suggested by Hafner[5] into one of the following groups:

Group 1. Fractures in which there is minimal displacement of the fragments (Fig. 1, *left*)

Group 2. Fractures in which there is some displacement of the fragments and also some comminution (Fig. 1, *center*)

Group 3. Grossly comminuted unstable fractures in which less than one third of cortical surfaces is left intact (Fig. 1, *right*)

In most instances all fractures falling in Groups 1 and 2 can be opened and fixed, any of the conventional angle plates being used.

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FIG. 1. *(Left)* Group 1. Roentgenogram of a nondisplaced stable intertrochanteric fracture which can be treated by a nail and a plate. *(Center)* Group 2. Here there is more displacement and some comminution but enough cortical surface to allow of positive internal fixation. *(Right)* Group 3. Marked comminution of the fracture, which makes correction of the varus deformity by internal fixation very difficult.

Occasionally more comminution is found at the time of operation than was anticipated, and difficulty is experienced in obtaining good fixation. In these circumstances, rather than accept inevitable failure, it has proved to be expedient to perform an osteotomy at the level of the lesser trochanter and displace the shaft medially in much the same manner as a McMurray type osteotomy (Fig. 7).

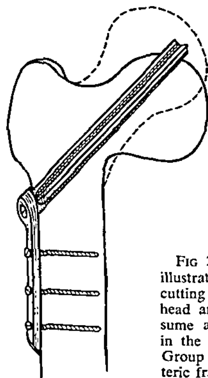


FIG 2. Diagrammatic illustration of the nail cutting through as the head and the neck assume a varus position in the comminuted, or Group 3, intertrochanteric fractures.

The treatment of Group 3 by open reduction and fixation is contraindicated, as the poor fixation obtained is inadequate.[3]

Similarly, to treat this group with traction will also result in the fractures healing in varus, as a traction apparatus will not differ from the angle plate fixation in preventing the proximal and the distal fragments from assuming bony contact, as previously described.

To cope with this type of fracture (Group 3) Bartels[2] advocates a method in which a valgus position is obtained that provides for good stability with pressure contact of the bone fragments. This is secured by a nonsurgical subcapital shaft transposition through the application of force that will shift the shaft medially in relation to the proximal fragment in much the same way as a high osteotomy. Occasionally this occurs as a result of the original injury and is referred to as "the reverse intertrochanteric fracture" (Fig. 3). The fragments assume

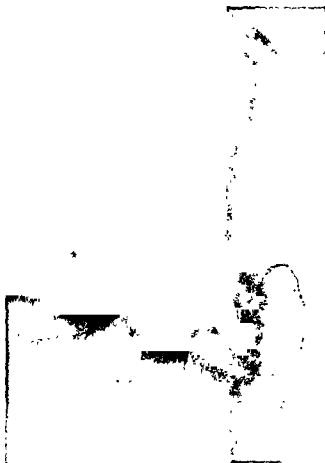


FIG. 3. This is the reverse type of trochanteric fracture in which the initial trauma transposed the shaft medially much in the same way as a Bartels osteoclasis.



FIG 4. Group 3 fracture which assumed a varus position and in so doing caused screw to break.

the same position as a subtrochanteric osteotomy.[7] The operation is performed in the following manner:

The patient is placed on a fracture table and given general anesthesia. The involved extremity is abducted widely, and traction is applied. Maintaining this position, force is applied to the lateral side of the femoral shaft just below the site of the fracture. Some spicules of bone will be broken off and crepitation obtained as the shaft shifts medially. A plaster spica then is applied. The patient may become ambulant with the aid of crutches in a relatively short period of time (Figs. 5 & 6).

Because no over-all procedure can be devised that will answer the many problems of each individual, the usefulness of traction[6] should not be overlooked in the treatment of intertrochanteric fractures. This is especially true when there are associated multiple injuries. It provides a simple, expedient method for adequate reduction and immobilization of the fracture allowing a greater leeway in treatment of other injuries.

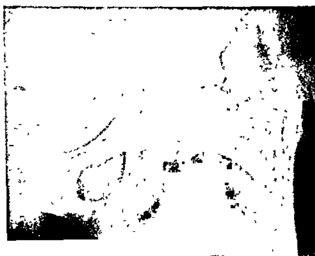
In reviewing the literature it was difficult to establish what the expectant mortality rate would be in the various decades of life. Such comparative figures would be valuable in evaluating the merits of any specific approach in the treatment of intertrochanteric fractures.

A total of 160 consecutive admissions of this fracture, covering a period of 5 years, were evaluated to determine the survival rate: 118, or 73.75 per cent, were females; 42, or 26.25 per cent, were males The figures are based on the hospital mortality, regardless of the service to which they were admitted or the treatment that they received.

Seventeen of the patients in this series were in the third and the fourth decades of life As would be expected, the fractures re-



FIG. 5. *(Left)* Group 3 comminuted intertrochanteric fracture. *(Center)* Position obtained by Bartels osteoclasis. *(Right)* Eight weeks later valgus position has been maintained and there is beginning osseous union.



sulted from rather severe violence, such as falls from scaffolds and automobile accidents. There were no fatalities in this group. The expectancy of life at age 40 in the United States is 33.19 years, according to actuary tables compiled in 1951 by the Metropolitan Life Insurance Company.

Sixty-two of the cases were in the age group from 50 to 70 years and carried a hospital mortality rate of 12.9 per cent. Four of the 8 patients who expired had a



FIG 6. *(Top)* Group 3 comminuted intertrochanteric fracture *(Bottom, left)* After reduction, Bartels osteoclasis, shaft displaced medially and abducted *(Bottom, right)* Advanced healing, 2 months later, valgus position of head and neck maintained



FIG. 7. *(Left)* Group 3 fracture in which an attempt was made to use internal fixation. *(Center)* Because of the comminution, no fixation could be obtained, hence an osteotomy was performed. Position similar to a Bartels osteoclasis obtained. *(Right)* Advanced healing. Patient had excellent functional result.

history of having experienced a cerebral accident prior to the time that they were admitted to the hospital. The average age of this group was 69 years. The expectancy of life age 69 for 1951 was 11.84 years. Aronson[1] reported a mortality rate of 15.8 per cent among 139 patients with an average age of 72 years. Goldenberg[1] reported 100 consecutive cases of trochanteric fractures with an average age of 74 years and a mortality rate of 16 per cent.

TABLE 1. PERCENTAGE OF EACH GROUP IN RESPECT TO AGE OF PATIENT*

GROUP AGE	PATIENTS NO OF	1 GROUP	2 GROUP	3 GROUP
37-60	12	16.7%	66.6%	16.7%
61-75	40	22.5%	55 %	22 5%
76-99	108	9.3%	46.3%	44.4%

* Note that comminuted fractures occur most frequently in the older age groups.

TABLE 2. END-RESULTS OF SERIES OF 80 PATIENTS TREATED FOR INTERTROCHANTERIC FRACTURES

NO. OF PATIENTS	GROUP IN WHICH CLASSIFIED	PROCEDURE USED FOR REDUCTION	ANGLE OF NECK OF FEMUR TO SHAFT FOLLOWING OSSEOUS UNION 120°-130°	Less Than 115°	NONUNION OF FRACTURE	RESIDUAL SHORTENING OF EXTREMITY
15	Group 1 and Group 2	Open reduction with angle plate fixation	13—(86%)	2—(14%)	0	0"-½"
18	Group 3	Traction	5—(28%)	13—(72%)	0	¾"-1¼"
11	Group 3	Open reduction with angle plate fixation	2—18 5%	9—81.5%	0	¾"-1¼"
36	Group 3	Bartels osteoclasis	23—63 9%	13—36.1%	1—.02%	½"-1¼"

The remaining 80 cases were in the age group of 76 to 98 years, the average age being 83 years. The expectancy of life at 83 years is 5.72 years. With the advance of the expectancy of life it will follow that a greater number of patients in this age group will be encountered. The geriatric problems concomitant with the patients in the eighth and the ninth decades are multiple. A good percentage of these patients were semi-invalids in precarious physiologic balance prior to the injury; others were remarkably well preserved. For instance, the oldest patient in this series was 98 years old when she sustained an intertrochanteric fracture classified as Group 2. Subsequently her fracture healed, and she was alive 3 years later. But, in general, the mortality rate of this fracture in the aged is high.

Of the 80 patients in this group, at the end of the first week of hospitalization, 8, or 10 per cent, had expired, the average age being 84 years. This group included 2 patients who sustained the fracture during confinement in a nursing home; another was the victim of an automobile accident.

At the end of the first month of hospitalization, 15 more, or 18.7 per cent, expired, the average age being 84 years. Of this group, one sustained the injury while a patient in a nursing home. Seven of them had signs and symptoms of cardiac decompensation on admission.

Eight more, or 10 per cent, of the group, with an average age of 85 years, had expired at the end of the third month of hospitalization. Of this group, 4 patients had clinical symptoms of cardiac disease on admission.

SUMMARY

1. Intertrochanteric fractures should be classified according to the degree of comminution present.

2. A Bartels closed osteoclasis is an ideal procedure for the treatment of comminuted intertrochanteric fractures.

3. A good percentage of these fractures occur in the seventh and the eighth decade of life and carry a high mortality rate. The geriatric problem is predominant, and positive treatment of the fracture alone is not sufficient to ensure a good end-result.

REFERENCES

1. Aronson, H.: Treatment of intertrochanteric and pertrochanteric fracture of femur. Traction treatment or internal fixation? Acta. chir. scandinav. 100:100-129, 1950.
2. Bartels, W. P. (Hempstead, N. Y.): Personal communication.
3. Evans, E. M.: Trochanteric fractures; review of 110 cases treated by nail-plate fixation, J. Bone & Joint Surg. 33-B:192-204, 1951.
4. Goldenberg, R. R., and Santaro, A. J.: Conservative treatment of trochanteric fractures, Bull. Hosp. Joint Dis. 12:27-40, 1951.
5. Hafner, R. H. V.: Trochanteric fractures of the femur, J. Bone & Joint Surg. 33-B: 513-516, 1951.
6. McElvenny, Robert T.: Reflections and opinions regarding the present status of fracture treatment, Clinical Orthopaedics No. 2, pp. 20-26, Philadelphia, Lippincott, 1953.
7. Wright, Louis T.: Oblique subcervical (reverse intertrochanteric) fracture of femur, J. Bone & Joint Surg. 29:707-710, 1947.

Fracturas Intertrochanteric

Summario in Interlingua

Le autores presenta un concepto pro le tractamento de fracturas intertrochanteric que ha provate se satisfactori in le practica. Le fracturas es classificate in tres categorias secundo le grado de comminution al sito del fractura. Le rationes que justifica iste classification es discutite.

Es describite un methodo que permitte le conversion de un comminuite fractura intertrochanteric instabile a in un stabile position

valge. Isto es effectuate per un osteoclase que move le fragmento distal a in un position simile al position obtenite per un osteotomia de McMurray. Es includite un summario tabular del resultatos final obtenite in un serie de comminuite fracturas intertrochanteric que esseva tractate per le methodo describite.

Cento sexanta casos de fracturas intertrochanteric es evalutate con respecto a etate, sexo, e typo de fractura. Es includite datos de mortalitate pro le serie total secundo le varie decennios del vita, sin reguardo al servicio hospitalari a que le patientes esseva admittite e sin reguardo al typo de tractamento usate.

24

The Immediate Treatment of Intracapsular Hip Fracture

ROBERT T. MCELVENNY, M.D.*

INTRODUCTION

There is a category for each fractured hip. The terms used here, such as "correct case" and "ill-chosen case," do not imply a method of eliminating any fractured hip from treatment. These terms do imply the proper designation of each intracapsular hip fracture in relation to the method of treatment chosen for that particular fracture.

Failure to choose the proper case for pinning, failure to obtain proper reduction or failure to place the fixation correctly lead to nonunion or delayed union between the femoral neck and head.

Closed pinning of an intracapsular hip fracture usually leads to immediate bony union, provided that the correct type of case is chosen and the proper reduction is achieved.

Occasionally, in these firmly united cases, aseptic necrosis of the femoral head will develop to the point of interfering with hip function. This complication seldom is encountered, and, ordinarily, a normally functioning hip is expected and obtained.

In certain cases osteotomy is indicated at the time of fracture, and as soon as possible in cases of nonunion or delayed union. Metal prostheses have no place in the treatment of intracapsular hip fracture if there is any chance of restoring any patient, at any age, to anything but permanent bed, chair or cane life. There is no substitute for a well-united femoral neck fracture. The attitude of not accepting nonunion or delayed union in the treatment of intracapsular hip fracture is, in our experience, well-founded.

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GENERAL CONSIDERATIONS

SPECIFICITY OF CERTAIN GRAFTS

Specificity of cells must be maintained if specific function is to be preserved. Liver cell must remain liver cell, corneal cell must remain corneal cell, skin cell must remain skin cell if the specific function of the part is to continue. Replacement of any specific cell is done by substitution. The substituting tissue cell is not specific and does not carry out the function formerly assumed by the specific tissue cell.

A specific cell has the power within itself to fulfill its destiny as long as its internal and external environments are within its limit of tolerance. Beyond this tolerance the cell is forced to modify. Once the cell loses its specificity it fails to propagate itself in kind, thus causing physiologic death. The area formerly supplied by this cell loses its ability to carry out its destiny, substitution occurs, and the specific function of the part is lost.

The internal environment of a cell may change its destiny. Virus or bacterial invasion of a cell may modify or kill the cell's normal destiny, and embolism, from solid or gas, may do the same. Abnormal heat or pus

may coagulate protein and kill the cell. Crushing or severe shock to cells at the time of injury, or as a result of treatment, may deprive the cells of their power to propagate in kind. Many other factors that affect the internal environment of cells could be mentioned to show that at present only a few are under our control.

Lack of metabolites, through denying external nourishment beyond the normal tolerance of the cell, will cause physiologic death. This fact is closely related to the type of tissue, the degree of metabolic demand, the temperature at which this demand is made and the functional demands made upon the part.

Shaft bone lying between the epiphyseal plates has generalized functions. Substitution of one portion of this bone for another throughout the body is possible. Following the substitution, this shaft bone ultimately blends and modifies to the part, so that in size, shape and form it assumes an appearance that is practically normal for bone in the area where it is placed.

A cancellous epiphyseal bone end comprising a portion of a joint seems to be more specific in nature. The epiphyseal bone arises from germinal centers that are not directly related or connected in origin to shaft bone. In youth this epiphyseal bone nourishes and stimulates the cells of the attached cartilaginous epiphyseal plate. The epiphyseal cartilaginous plate does not contribute to the growth of the bony epiphysis. Throughout life the epiphyseal bone supports and nourishes the articular cartilage upon its surface.

All grafts, in order to function properly, must maintain their relative amount of specificity. A graft of an orange bud to a lemon tree produces oranges, not lemons. A skin graft must produce skin rather than scar, if it is to succeed. The femoral head is a specific graft. If it is to remain femoral head it must live. If it dies, the replacement tissue is nonspecific and will not carry out the form, the shape or the function of its predecessor.

In the case of the femoral head, seldom can one control the crushing, the shock or the infection that initiates the internal environmental factors. The fate of some heads is sealed at this time. Further, some femoral heads are dead at the time of fracture, and some dead heads seldom, if ever, suffer fracture at the neck (malum coxa senilis, Caisson disease, etc.).

Fortunately, most femoral heads separated from their necks are living, specific tissue. The cells of a femoral head, like bone elsewhere, have certain powers of hibernation. The object of treatment is to provide a normal external environment for these cells as quickly as possible. Solid bony union is the normal condition at the fracture site. This must be established quickly throughout the *entire fracture surface*. It is evident that if only a portion of the neck contacts the head, the portion of the head not supplied by this contact suffers from inadequate nutriment. When complete fracture surface contact has been obtained and immediate bony union ensues, the normal blood flow pours through the fracture site to the head from the trochanteric and neck pools. However, if the normal external environment at the fracture site is not provided within the tolerance of these specific bone cells, these cells eventually die and are not reproduced. Creeping substitution ensues, and thus the integrity of the femoral head is lost. Nonunion and delayed union at the fracture site produce such effects.

An example of the loss of specificity can be seen when a femoral head that has undergone aseptic necrosis is bone-grafted or osteotomized so that bone replaces by creeping substitution. The femoral head so treated never again becomes a normal head. Subchondral bone and the articular cartilage degenerate, shape is not restored, and eventually arthritic changes develop. The same loss of cell specificity is seen in certain




FIG. 1 A. Actual specimens of intracapsular hip fractures. A subcapital type is shown on the left, a transcervical type is shown on the right.

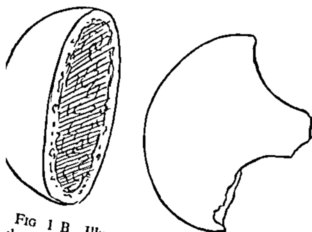
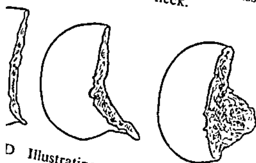


FIG 1 B Illustrating the two types of ıbcapital fracture. If a large bone mass ›tains on the superior portion of the ad, this mass will prevent the moving the shaft medially because this mass ; its counterpart on the neck.



D Illustrating types of transcervical neck fracture.



FIG. 1 C. Subcapital fracture shown on the right in Figure 1 B. Other type of subcapital fracture is shown in Figure 17, top.

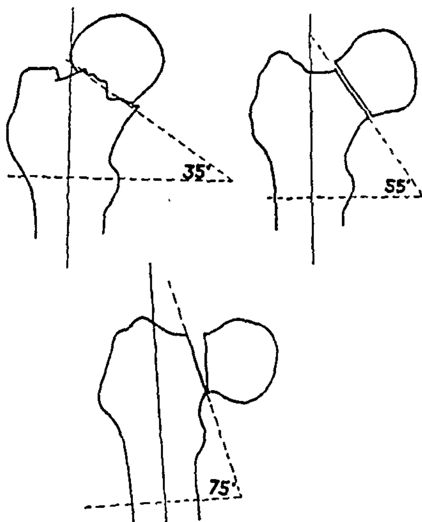


FIG. 2. Linton's lines to show the significance of obliquity of fracture planes. Note that the first figure shows an adequate reduction. Fortunately, fractures do not occur in flat saw-cut planes. Compare with Figures 3, 4, 7, 12, 15 and 16. (Dickson, J. A.: J. Bone & Joint Surg. 29:1006)

cases of slipped upper femoral epiphysis. The resultant healed case loses its normal articular surface, contour and function.

The above contentions have been held by this writer for 20 years. He has insisted that most dead femoral heads are produced by indifferent treatment—that the temporary loss of the external blood supply to the head is relatively unimportant. He has shown femoral heads alive and functioning 20 years after femoral neck fracture and 15 years after union finally was obtained at the fracture site. On many occasions he has demonstrated that the "latent life" or "hibernation" of bone cells occurs in the femoral head of the human. Femoral heads considered dead by roentgenographic standards and by people of established reputation have been united to their necks and over the years continue to maintain shape, joint space and function.

This writer has cited the improvement in the results obtained in the treatment of oblique fractures of the lower third of the tibia and in the fracture of the carpal navicular. Improved methods of treatment have done this. Not many years ago, the lack of external blood supply to the tibia or the carpal navicular was given as the cause of the poor results obtained. At that time the carpal navicular was compared with the head and the neck of the femur in explaining the failure of union in the navicular and the consequent disintegration of the bone. The methods of treatment of these fractures have changed, and the results have improved beyond expectations. It is patent that, while the methods of treatment have changed, the conditions at the fracture sites have remained constant.

Dr. E. J. Tucker, in a personal communication, states the following:



FIG. 3. "Beak" (malignant) type of neck fracture. This type is easily reduced and is easily united. *(Top, left)* Shows an inadequate reduction with the head fragment medial to the neck. *(Top, right)* Eight months after pinning and 2 months after weight-bearing. Delayed union now is evident and is shown by the proliferative callus mass at the fracture site pointed to by the arrow. Osteotomy is indicated at this time. *(Bottom)* Eight years after pinning. Solid bony union is present but has arrived too late to save the femoral head. This is a complete failure because of the arthritic head changes. (Compare these views with Figs. 6, 12 & 15.) (McElvenny, R. T.: S. Clin. North America 37:253)

Bone cells are capable of existing almost indefinitely without an independent blood supply. One of the most common examples of this is the fact that a piece of bone may remain free in a joint cavity for many years and the osteocytes will remain intact and alive. I have kept cortical bone in its own plasma for as long as two years and then placed it in the subcutaneous tissue of a rat. The bone cells would absorb their own body matrix and revert back to connective tissue cells.

This was also reported by Dr. L. A. Peer in *Transplantation Bulletin*, vol. 1, no. 1, September, 1953.

Choosing the Hip Fracture for Closed Reduction and Pinning

Whether or not a fracture unites depends mainly on the relationship of the bones at the fracture site. Absolute anatomic reduction is usually the ideal in spiral fractures of shaft bones. Compression forces are applied and maintained while correct fixation is accomplished. If this is done, union is rapid, but there is a small amount of callus production. The more motion there is at the fracture site and the poorer the apposition of

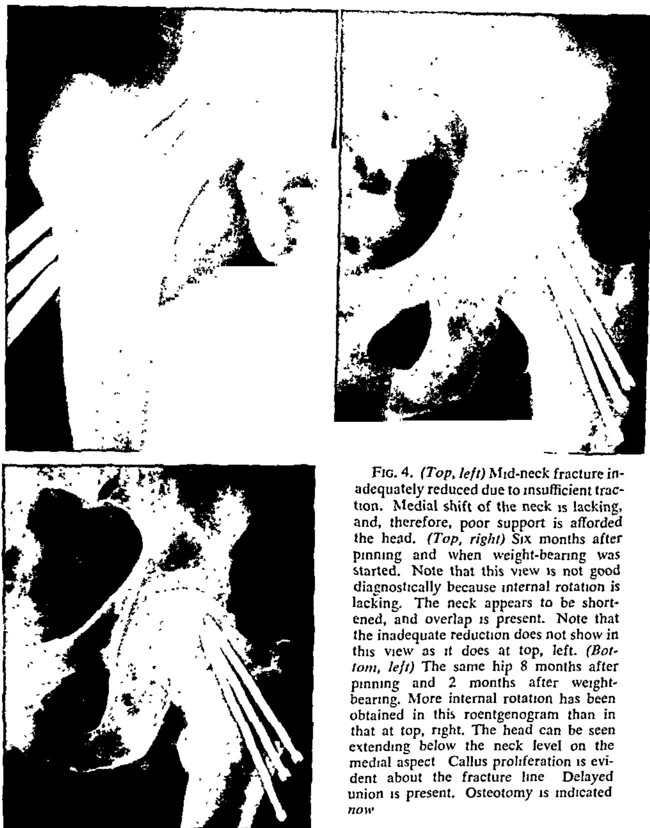


FIG. 4. *(Top, left)* Mid-neck fracture inadequately reduced due to insufficient traction. Medial shift of the neck is lacking, and, therefore, poor support is afforded the head. *(Top, right)* Six months after pinning and when weight-bearing was started. Note that this view is not good diagnostically because internal rotation is lacking. The neck appears to be shortened, and overlap is present. Note that the inadequate reduction does not show in this view as it does at top, left. *(Bottom, left)* The same hip 8 months after pinning and 2 months after weight-bearing. More internal rotation has been obtained in this roentgenogram than in that at top, right. The head can be seen extending below the neck level on the medial aspect Callus proliferation is evident about the fracture line Delayed union is present. Osteotomy is indicated *now*

the fragments, the greater the callus mass that is produced. However, other fractures often require modifications of bone by an osteotome, a graft or a gadget to obtain adequate surface contact with adequate compression and locking of the fragments.

The fracture through the femoral neck cannot be held in an anatomic position. The cortex of the femoral neck is so thin that one might as well try to appose the ends of

2 fingernails as to hold cortex to cortex at the junction of femoral head and neck. Following slight absorption of bone, the medial inferior cortex of the head either settles medial to that of the neck (bad) or lateral to that of the neck (good) (see Figs. 8, 10, 17 & 20).

An incorrect relationship is one in which the femoral neck fails to furnish support to the medial inferior portion of the femoral head. Because of this lack of support and the slanting of the fracture surface, the muscle pull and the thrust of the body on the fractured bones allow full angulation and shear forces to act at the fracture site. Adduction of the lower extremity causes angulation of the neck upon the head. The neck first distracts from the superior portion of the head and then tends to slip up and out and by the head. The head goes toward varus. Since there is no support of bone-to-bone, the full component of all unfavorable forces is spent upon the fixation material, the only support afforded the head in this situation. The result is a migration, bending or breaking of the fixation material (see Figs. 4 & 7).

A favorable relationship at the fracture site is one in which the medial portion of the femoral neck is moved to the inner and under side of the corresponding portion of the femoral head. Once this medial shift of the femoral shaft actually is obtained, it is maintained by the correct insertion of the fixation material. The fixation material guides the fragments to their predetermined situation. Bone supports bone. All components of thrust are spent against this bony buttress of the neck. The elimination of shear and angulation forces by this locking of fragments alleviates the subtending force that otherwise would be applied to the unsupported head by the fixation material (see Figs. 8 & 15).

The soft cancellous bone of the femoral neck is confined by a thin cortical shell. The medial portion of this cortex forms a V-shaped mass with its apex facing the pelvis. It is approximately ⅛ of an inch thick and about 1 inch wide. This area is often spoken of as the calcar femorale. The calcar slants medially from below upward. It is the strongest and thickest portion of the femoral neck because it is in line with and receives much of the component of forces placed upon the femoral head and neck. As the femoral head is approached, the calcar femorale fans out, becoming flatter, and in a gentle, graceful curve blends with the larger and overlapping femoral head (see Figs. 9 & 10).

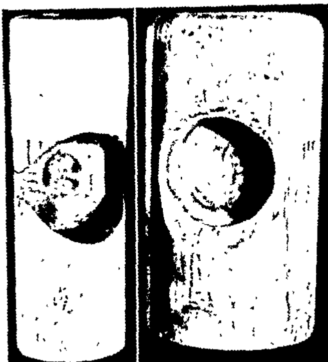


FIG. 5. The perineal post on the left is the correct size; that on the right is too large. Cutouts are for the roentgenographic head attachment.

The femoral head may be cleanly snapped off the junction with its neck, leaving no portion of the *calcar femorale* or cancellous bone protruding from its surface. This clean break occurs at about the level of the former epiphyseal plate. This fracture resembles in location and in kind a slipped femoral capital epiphysis of youth. *This type of fracture is defined as a subcapital neck fracture.* By definition, *no cancellous or cortical portion of femoral neck bone remains firmly attached to the lower half of the head.* This

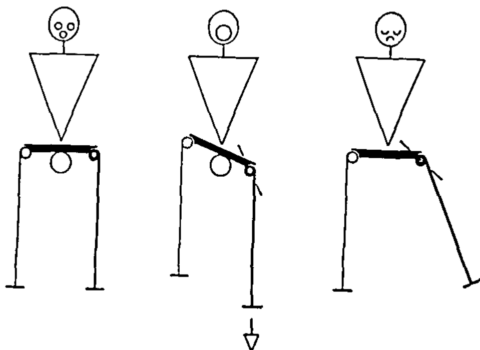


FIG. 6. *(Top)* Shows the effect of not fixing the pelvis by traction on the normal limb before severe traction is applied to the affected side. If this error is made, the slanting pelvis produces a relative abduction of the limb. In these circumstances an adequate reduction is virtually impossible.





FIG. 6. *(Bottom, left)* "Beak" type fracture. Traction is severe. Medial displacement of the shaft has been attempted repeatedly, but adequate reduction is not obtained, as shown here. Inspection of the patient showed a marked shift of the pelvis to the affected side. To meet this situation, all traction was released from the affected limb. The patient was squared onto the perineal post so that the perineum centered the post. Additional traction was applied to the normal limb Reduction then was attempted For the result see right. *(Bottom, right)* Reduction now is adequate Note the center post in relation to the symphysis in bottom, left and right There is a gadget that keeps the pelvis level under unilateral heavy traction and still allows full operative freedom. This apparatus fits almost any orthopaedic table

FIG. 7. *(Top, left)* This reduction is anatomic. Note the adduction of the shaft and the complete internal rotation. However, medial displacement of the shaft is lacking, due to insufficient traction. *Note that the guide wire does not enter deep into the acetabulum. The head is not in varus.* On the medial surface of the neck, lines have been drawn upon the wet spot film. These lines show that the reduction is inadequate. Compare with Figure 13, top, left. *(Top, right)* The nail has been driven home. The reduction remains inadequate. Lateral view is shown in Figure 21. *(Bottom, right)* The result a few weeks later. The head falls to varus, and the metal cuts its own channel in the cancellous bone of the head. Note the "beak" of bone on the inferior medial portion of the head that could have been caught and locked to the neck by an adequate reduction. Now osteotomy following traction and repetition of the reduction are indicated. (McElvenny, R. T.: S Clin North America 29:43)



type of fracture is uncommon before the age of 60 but increases in frequency as age advances.

The other type of femoral neck fracture is one *in which a portion of the neck, either cortical, cancellous, or both, remains firmly attached to the inferior half of the femoral*



FIG. 8. *(Left)* Specimen of a fractured hip that was never treated. This specimen has been cut in half, as the only cortical projection on the head was in the posterior half. This beak is small but strong. Note its shape. If complete internal rotation with medial displacement of the neck is not obtained at reduction, the neck will not get under this projection on the head. Then this head spicule will not contact the neck, and thus invagination and locking of the head to the neck will not occur. Next to the middle finger the posterior portion of the neck in the region of the *calcar femorale* is shown This again emphasizes the importance of medial displacement and complete internal rotation of the shaft



(Right) This view shows the thickness of the lower border of the neck A slightly overreduced (adequate) position is demonstrated, but it is still practically anatomic. The head could slip off the neck, but the tendency here should be for the head spicule to fall into the cancellous portion of the neck. *(Bottom, left)* If the head spicule fails to fall into the cancellous portion of the neck, the result will be as shown here.

(Continued on following page)

head. This type is known as a transcervical fracture. The actual viewing of a transcervical fracture shows not a straight line of fracture but an irregular surface first insulted by a separation of the head from its neck in the upper portion, and then, as the head is bypassed by the neck, the head takes with it, by ripping and avulsion, some bone that is normally neck property. This leaves upon the inferior half of the femoral head a V-shaped mass of cortical or cancellous bone. The apex of this funnel-shaped bony mass



FIG. 8 (*Cont.*). (*Left*) If the head spicule falls into the neck cavity, locking and capping of the head upon its neck will occur as illustrated. (*Right*) To avoid minimal reduction and doubt, plenty of traction, medial shift and internal rotation will produce the ideal situation, as shown here. Note that upward thrusts are converted into impacting forces, and note how much of these forces falls to the inner side of the head fragment. (Compare with Figs 12, 15 & 16)

points toward the neck. *The object of reduction is to take advantage of this fortuitous anatomic circumstance* (see Figs. 8, 9, 15, 17 & 20).

When the femoral shaft actually is shifted medially, a point is reached where the neck mass of bone attached to the head will completely invaginate the soft cancellous sheath of the femoral neck. This is practically an intramedullary graft of the head to the neck. The cortical shell of the neck cups and supports this mass and the attached head in symbiotic bliss. The most important area of support thus gained is in the region of the *calcar femorale*. However, due to the medial shift of the femoral neck, the superior lateral portion of the femoral head overhangs the cortical surface of the neck. This allows the femoral neck to gouge into this cancellous portion of its head and results in locking by the head and the neck at two main points, cortex to cortex (see Figs. 8, 9, 12, 17, 19 & 21).

Thus, angulating and shearing forces are converted into thrusts that compress bone against bone. The greater the thrusts, the deeper the invagination and the locking of the fragments. The acetabulum above and the femoral neck below cause a "swedging" to occur at the fracture site because it is practically impossible for the femoral head to escape from its neck. It does not matter how much of the neck remains on the femoral head *so long as enough remains in the lower half to allow invagination and engagement of the fracture surfaces.* Once engagement of the head and the neck is correct, it is of no moment what angle of fracture one started with. The engagement and the locking eliminate shear and angulation and convert these into compression forces. The compression force varies the angle to the most favorable situation possible by squeezing the inner portion of the head and its attached neck against the *calcar femorale*, which, in turn, levers and maintains the head upon its neck. The superior lateral portion of the femoral head then must settle



FIG. 8. *(Left)* Specimen of a fractured hip that was never treated. This specimen has been cut in half, as the only cortical projection on the head was in the posterior half. This beak is small but strong. Note its shape. If complete internal rotation with medial displacement of the neck is not obtained at reduction, the neck will not get under this projection on the head. Then this head spicule will not contact the neck, and thus invagination and locking of the head to the neck will not occur. Next to the middle finger the posterior portion of the neck in the region of the *calcar femorale* is shown. This again emphasizes the importance of medial displacement and complete internal rotation of the shaft.



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FIG. 8 *(Cont.)*. *(Left)* If the head spicule falls into the neck cavity, locking and capping of the head upon its neck will occur as illustrated. *(Right)* To avoid minimal reduction and doubt, plenty of traction, medial shift and internal rotation will produce the ideal situation, as shown here. Note that upward thrusts are converted into impacting forces, and note how much of these forces falls to the inner side of the head fragment. (Compare with Figs. 12, 15 & 16)

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Thus, angulating and shearing forces are converted into thrusts that compress bone against bone. The greater the thrusts, the deeper the invagination and the locking of the fragments. The acetabulum above and the femoral neck below cause a "swedging" to occur at the fracture site because it is practically impossible for the femoral head to escape from its neck. It does not matter how much of the neck remains on the femoral head *so long as enough remains in the lower half to allow invagination and engagement of the fracture surfaces.* Once engagement of the head and the neck is correct, it is of no moment what angle of fracture one started with. The engagement and the locking eliminate shear and angulation and convert these into compression forces. The compression force varies the angle to the most favorable situation possible by squeezing the inner portion of the head and its attached neck against the *calcar femorale*, which, in turn, levers and maintains the head upon its neck. The superior lateral portion of the femoral head then must settle



FIG. 9 A. Illustrates a true anteroposterior view of femoral head and neck. Note the position of the lesser trochanter. Only this view, with traction, and head and neck separated, is of diagnostic value in considering the type of fracture and the adequacy of the reduction. A roentgenogram without the above features has no significance.





FIG. 9 B *(Left)*. A hip fracture that is a few hours old This view of overriding and external rotation of the shaft would indicate a garden variety of hip fracture.

FIG 9 C *(Right)*. Under traction and internal rotation head and neck are thrown clear of each other and stand in bold relief. The condition at the fracture site now is clear but is not of garden variety. This emphasizes the value of the view advocated here

(Continued on following page)

further into the lateral surface of the neck. This mechanism must continue until the femoral head caps its neck by shifting to receive directly the component of all thrusts in line with weight thrust and muscle pull. The head is locked firmly and "swedged" into position, and compression forces now are working correctly on certain areas of contact, regardless of the obliquity of the fracture one started with (see Figs 9, 12, 17, 19 & 21).

THE ANGLE OF FRACTURES

In an inadequately (under)reduced or in an anatomically reduced intracapsular hip fracture, the more perpendicular and steep the line of fracture is, the greater the effect of telescoping, angulation and torque will be at the fracture site. This is due to the fact that there is no support of bone to bone, and all that holds the head from slipping downhill is the fixation material. Under these circumstances, the fixation material



FIG. 9 D (*Left*). A true lateral view of a femoral neck with the reduction anatomic. Note that a spicule has been cut on the head that is posterior in location. Compare with Figure 21.

FIG. 9 E (*Right*). The lack of complete internal rotation of the neck on the head is shown. Note that the neck is hitting the head, and more internal rotation will accomplish little unless distraction is accomplished by traction. The fracture is also underreduced, for the head fragment is medial to the neck fragment. Note that in this view the head tilts downward, and there is a projection of the neck above the head and of the head below the neck. Compare with Figure 21.

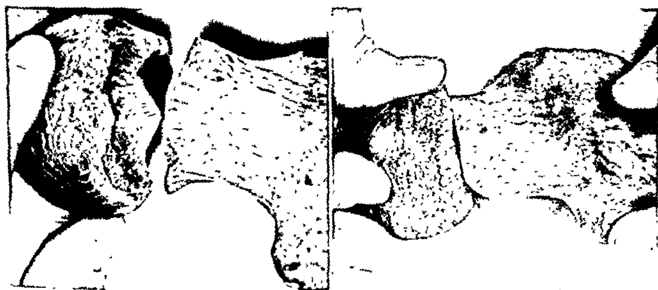


FIG. 9 F (*Left*). Overpull by traction is demonstrated. Complete internal rotation produces slight eccentricity between head and neck contact. Adequate reduction is shown here, for, in addition to *complete internal rotation*, medial displacement of the neck beneath the head has been produced Note alignment of head and neck.

FIG. 9 G (*Right*). The result of impaction in the position is shown in F. The neck now supports the head well, and the spicule on the head has invaginated into the neck and thus is hidden partially by the neck buttress. Note the alignment of head and neck and that no projection of the neck now extends above the head but that the reverse is true Compare with Figure 21.

must support and subtend the head, and since this is impossible, migration of the head and the metal ensues.

The obliquity of the fracture line at the hip is of interest and of prognostic significance only in (1) subcapital fracture, and (2) transcervical fractures that have been inadequately (under)reduced or put together anatomically. *The observations and the derived conclusions about the signifi-*

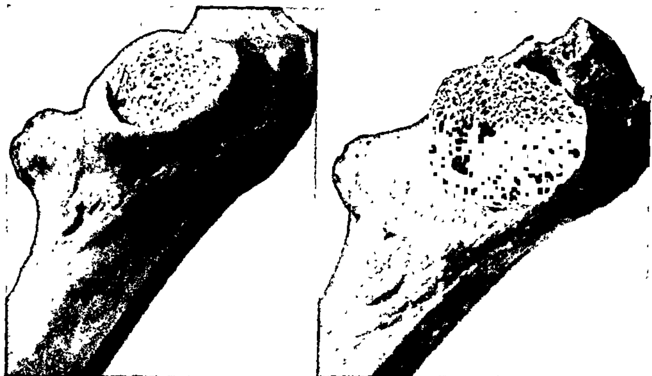


FIG. 10. Cut surfaces of femoral necks vary in shape and size and cortical thickness. In general, the superior and the inferior areas are the narrowest in diameter, the center diameter being the widest. This allows the spicule on the head fragment to invaginate easily because it is from the narrower portion of the neck.



FIG. 11. *(Left)* This type of reduction is not uncommon. A lateral view would show that reduction is inadequate If more traction had been used, the neck could have been slipped under the head. The neck has not been overpulled; thus, it has hit the head upon internal rotation and, therefore, has been blocked from internal rotation by hitting the head. Close inspection of this anteroposterior view shows that the neck lies anterior to the head. Locking is impossible because, since the neck hits the spicule on the head, it can never get under the head for invagination *(Right)* The result a few months later. Now it is evident that the spicule on the head has *always* been behind the neck. This failure in spite of the medial displacement, because insufficient traction was used and head and neck did not clear each other Osteotomy now is indicated



FIG. 12. *(Left)* Contrast this comparable view with Figure 11, left. Note here how the neck is under, and not anterior to, the head. Note the plain evidence of locking of the fragments. To get this neck under this head, severe traction and maximum rotation were necessary, because this is a long "beak" type of fracture. Note that more nail extends out from the shaft than penetrates into the head. In contrasting this view with that of Figure 11, left, note how both head and neck are in bold relief and no overlap is present. Note the overlap in Figure 11, left, because of the lack of anatomic line-up. *(Right)* Solid bony union is present The nail has not shifted, although it is only a comparatively short distance into the head. This nail would have shifted if locking of bone to bone had not been present This demonstrates the slight angulating and telescoping forces placed upon the fixation material in properly reduced and, therefore, properly supported, femoral neck fractures. This is a view of the left hip of the patient shown in Figure 18. (McElvenny, R. T : S. Clin. North America 29:47)

cance of these angles at the fracture line have been made and derived from the above type of data and standards. These statements and conclusions as to the effect of obliquity of the fracture line *have no significance* as regards prognosis when applied to transcervical fractures in which proper reduction and fixation are achieved.

It is evident that if both levers forming an angle are freely movable in all directions at the point of intersection of the levers, no angle need remain constant. In dealing with transcervical hip fracture, it is possible to overpull and shift the neck to a point under the head where locking of the fragments occurs. In this situation the thrusts are bone to bone and not bone to metal to bone. The metal serves only as a guide and prevents external rotation of the neck upon the head. If the latter should occur, the fragments will be disengaged from their locked position (see Figs. 2, 8, 9 & 20).

BONY UNION

It is not difficult to reduce a hip. It is not difficult to do an osteotomy. It is difficult at times to keep in mind what makes fractured or grafted bone fulfill its destiny and unite. For years man has known that, all other things being satisfactory, bone tends to unite if torque (rotational), shearing (telescoping) and angulating forces are eliminated. However, the elimination of these factors is of little value unless at the same time all available bone surfaces are apposed to one another and squeezed physiologically into firm and intimate contact by *a constant com-*

FIG. 13. *(Top)* Lines similar to these shown are used on the wet spot roentgenograms to indicate inadequate or adequate reduction. Figure 7 illustrates this point. On the left an adequate reduction is shown, and on the right an inadequate reduction is demonstrated. Impacted fractures show the same situations, provided that the roentgenogram is taken with no lesser trochanter visible in the film

(Bottom) Here is an impacted intracapsular hip fracture which, on the left, shows a roentgenogram taken in external rotation This roentgenogram is not of diagnostic use. On the right a similar view is shown with the limb rotated internally. Note that both head and neck are in clear relief, and all criteria of adequate reduction obtain. This is a so-called abduction type of impacted hip fracture Reduction is not necessary; all one has to do is pin it. The adduction, or inadequate, type must be pulled apart and reduced until it achieves the situation shown in this illustration If, after viewing either type of impacted fracture in the correct position, a subcapital type is encountered, osteotomy of the McNeur type is the immediate treatment of choice. Contrast this with Figures 11 and 12



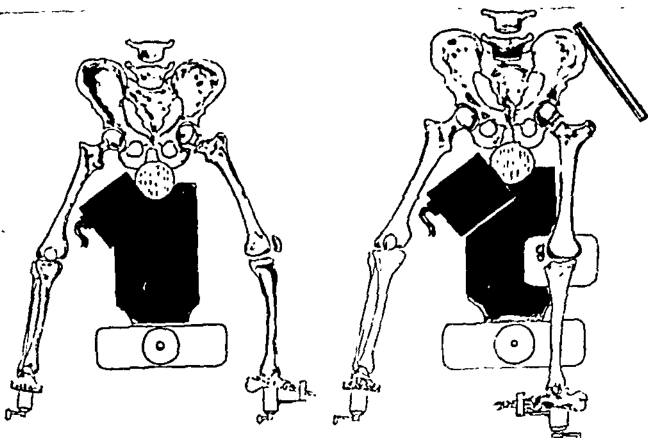


FIG. 14. *(Left)* Schematic drawing showing a level pelvis and traction to the normal limb. Note that complete external rotation of the affected limb is demonstrated and that abduction obtains. The clearing of the head by the neck is shown plainly. Because of this clearing by traction, complete internal rotation and medial displacement are possible. Note the position of the roentgenogram tube. Note the traction on the normal limb.

(Right) From a position of external rotation and abduction the shaft now has been completely rotated internally and adducted. Note the position of the foot and the knee. Note that distraction is present. The hip is maintained in this position by the locking of the table, and fixation now is completed with no disturbance of this situation until the procedure is completed and proved to be so by roentgenographic views. Compare with Figures 15 and 16. (McElvenny, R. T.: S. Clin. North America 29:34 & 36)

pression force directed at right angles to the line of fracture or the line of apposition where union is desired. The most potent factors supplied by the body to furnish compression forces are muscle pull, tissue weight and body thrust. It is obvious that unless these components work upon bone surfaces correctly, thereby more firmly locking or pressing together the broken ends or apposed surfaces, nothing is gained except an increase in the effects of torque, telescoping and angulation.

Metal fixation is essential at times. Be it wire, screw, band, plate or rod, the compression force at right angles to the surfaces that are to unite must be the dominant force along with the most intimate contact possible between the fragments.

A hip pin is only a metal rod along which the head and the neck glide to be compressed and locked together in intimate contact by muscle pull, tissue weight and body thrust Unless the femoral head is locked to its neck and the compression forces are at right angles to the area where union is to take place, failure results. A correctly performed osteotomy and a correctly reduced hip presume the shifting of both the femoral head and the femoral neck in order to obtain this ideal situation.



FIG. 15. *(Top, left)* The fracture at admission. The shaft is in external rotation, and overlap is evident between head and neck. Further, one cannot say whether the "beak" is or is not fractured away from the head. From views like this it is impossible to tell the type of fracture or, very often, the condition of the femoral head. See Figure 9 A-C and 16, top, left.

(Top, right) This view was taken under severe traction, and complete internal rotation throws head and the neck into complete and bold relief. Reduction now is adequate, and invagination is possible. The "beak" is attached firmly to the head fragment. From this view, and this view alone, can one determine what the fracture is. This view is of true diagnostic significance. At this time one determines to pin a true transcervical type or to osteotomize a subcapital type.

(Bottom, left) Four days after pinning. Note the slant and the depth of the nail. The medial side of the head *has deeply invaginated the neck.* On the lateral side *the neck has invaginated the head fragment.* Firm locking has been achieved, and all components now will be favorable for union.

(Bottom, right) Four months after fixation. Laterally and medially invagination is evident. The nail has not changed its position. Weight-bearing can start now.





FIG. 16. *(Top, left)* The fracture at admission. From this view it is impossible to determine accurately the amount of neck on the head or the true type of fracture. External rotation of the shaft is present. The head has been spun so that its fracture surface looks to be somewhat posterior. *(Top, right)* Severe traction, complete internal rotation, medial shift and adduction show that reduction has been achieved. Note the clear relief of head and neck. This is a midneck fracture with plenty of bone on the medial and the lower portion of the neck. It is evident that the proposition of having the head invaginate the neck, which constitutes an adequate reduction, obtains here.

(Bottom, left) The nail is at the correct position and depth. Note that the guide wire is deep in the acetabulum to fix the head so that it will not shift or turn as it accepts the nail. Severe traction still is maintained. Distraction between head and neck remains. No impaction is necessary or desirable. *(Bottom, center)* Six days after pinning. The head has settled well on the waiting neck in correct relationship. The situation now is well in hand, and union is predictable. The nail is set deep, but has been run out and in a few times, so that it slips easily. This is done so that, if too deep, it automatically extrudes to the exact minimum to clear the acetabulum. All that is necessary is to prevent external rotation of the neck on the head. The locking of the fragments subtends the head on the neck. The nail is placed deep, as tipping of these long neck fractures without a marked "beak" is surely a tendency. *(Bottom, right)* Four months after pinning. Note bone beneath nail at fracture junction. No shifting of the nail has occurred. Weight-bearing was started at this time, the patient using only a cane.





FIG. 17. *(Top)* This is a typical subcapital type of fracture. Note the distraction and the diagnostic quality of this view under severe traction and internal rotation. Note that on the upper superior portion of the head there is a projection of neck *that prevents the medial displacement of the shaft.* This patient had a carcinoma of the urethra Intensive radiation was given with adequate portal protection. A year later she developed the fracture shown here.

(Bottom, left) Repeated attempts at overpull and medial displacement failed to gain an adequate reduction. It was thought that if the head could be slipped lateral to the neck, the projection on the upper outer portion of the head would lock the head to the neck. The capsule of the hip joint was opened anteriorly, the index finger spun the head to varus, the shaft was displaced medially and then the head was spun into valgus. The position obtained was fixed as shown here. This treatment of a subcapital fracture seldom is possible and is not advocated. *An osteotomy is routinely* a *better* choice.

(Bottom, center) Two years later the patient was walking normally without support. Suddenly she began to complain of stiffness and vague pain in the right groin. Until this time no complaint of the right hip had been reported. A roentgenogram taken at this time showed some varus as compared with roentgenograms of this hip taken 2 years before. Laminagraphic studies of this right hip then were made.

(Bottom, right) Laminagraphic study afforded two or three views that confirmed the intracapsular hip fracture. This again proves what the author has contended for 20 years—that an intracapsular hip fracture, in face of inadequate trauma, is a march or so-called "fatigue" fracture. In these people the hip is fractured days or hours before the actual disengagement. The external rotation separates the neck from the head, and these people fall because of this. They do not fall and then break the hip One view confirming the fracture is shown here

(Continued on following page)





FIG. 17. *(Left)* One year after fracture of the right hip and 3 years after fracture of the left hip. *Note how the left hip has settled in an almost anatomic position.* At time of pinning the right hip, the nail was removed from the left hip, as it was solid. Because of radiation therapy, bone grafts were placed to contact the upper outer quadrant of each head in an effort to stimulate the cells of these heads and inspire them to live. The common breakdown point in heads that are dead or dying at time of fixation is the upper outer quadrant. Here the full thrust of weight-bearing is received, and, therefore, functional and metabolic demands on these cells are greatest. It takes live cells to stand the demands. Note that on the right, slight varus now obtains, and the fracture line shown at bottom, right, page 224, is evident at the cortical break on the undersurface of the neck and also at the superior surface.

(Right) Seven years since the left hip fracture and 5 years since the right hip fracture. The patient lives a practically normal life without using external support. Occasionally, after a full day, she notices some vague ache and slight stiffness in the left hip. To date, this is of no consequence to her. She has no limp, no limitation of activity. The future of all hip fractures after 10 to 12 years is always debatable. Constant demands and stress following an injury of this sort, which, to start with, is often pathologic in some way, work on all tissue, including bone. Twelve to 15 years of normal living with a good solid hip beats the best substitute that we have now. Unite the hip and give it a chance to wear out. Do not unite it and it rusts out, because no life remains in the cells of the femoral head.

NONUNION

A well-reduced hip never changes its position. The metal does not shift. The fracture is locked in position, bone to bone. A reduced hip is practically painless from the time of pinning. Active control of the limb with complete internal rotation is present.

A lack of full internal rotation, pain in the groin, limp or some slight migration of metal indicate trouble and the need for definitive action. An osteotomy at this time produces bony union, while waiting produces greater deformity or complete disengagement (see Figs. 3, 7, 11, 12 & 16).

DELAYED UNION

Frank nonunion is evident, while delayed union is a subtle condition which, once understood, is easily recognized and, once recognized, can be turned into bony union by an appropriate osteotomy.

Delayed union is fibrous union at the fracture site. The head adheres to the neck, but slight motion is present. This motion causes refracture and irritation of the callus mass, which proliferates about the fracture site. The constant increase of this mass is picked up easily by serial roentgenographic studies.

If one deliberately placed cellophane between the fracture surfaces of the head and the neck of the femur, one would expect to have, following fixation, a routine disintegration of the femoral head. Delayed union produces a fibrous barrier to the normal blood supply of the femoral head, with-



out which the cells of the femoral head can live for only a limited time. The cells demand nourishment because the fixation material holds the head and forces it to function. *The demand on these head cells is far greater than if no attempt at reduction had been made, for then the disengaged femoral head lives as a loose body lives.*

Once delayed union is established, immediate bony union can be obtained by an osteotomy, with or without a bone graft. These procedures, performed early and correctly, ordinarily will give immediate bony union and will save the femoral head. *This*

FIG. 18. *(Top)* Photograph of the patient shown in Figure 12, 5 years after the "beak" type fracture of the left hip was pinned. When this photograph was taken the patient was 66 years of age and was enjoying normal hip function and normal activity. A few weeks later her right hip disengaged while she was running down the basketball court in play with some of her college students.

(Bottom, left) The right hip also shows a "beak" type of fracture. The reduction is adequate. *(Bottom, right)* The hips of this patient 13 years after fracture of the left and 8 years after fracture of the right. At this time there is no curtailment of activity, no pain and no limp Because the nail protruded so far, it was pulled out when the right hip was fixed.







FIG. 19. *(Left)* Three months after reduction and pinning. Note that internal rotation has put the neck under the "beak" of the head, but medial displacement is so slight that only a minimal chance of locking and invagination is possible. For a few more weeks one will not know what will happen. Which way will the "beak" on the head go? Will it catch on the cortex of the neck and lock? Will it slip to the medial side of the cortex of the neck and begin falling off or move around like a pill in a box and develop non-union? *(Center)* Six months later the patient disengaged the left hip. This was reduced with no doubt about reduction. *(Right)* A few days later this roentgenogram showed the left hip to be well settled on the neck in the correct position. The right hip fell and locked to the neck and is now solid. The left hip is predictable and will be solid also. The patient has equal and full control of both hips at this time. The nail is not deep enough but will be adequate and will not shift. Note the almost identical position of both heads as to valgus and position upon their respective necks.

one fact is the most important single consideration in anyone's conception when dealing with fractured necks of femurs. Delayed union can be picked up within the first 8 months following fixation. Usually femoral heads can be saved within a year of the time of original fixation (see Figs. 3 & 4).

Valgus and Varus

Valgus and varus are terms which describe a fixed attitude at or near a joint If the apex of the angle of the two levers forming the joint while in the anatomic position point toward the mid-line of the body, a valgus position obtains. If the apex of the angle points away from the mid-line of the body, a varus position obtains.

For example, the elbow's normal carrying angle is a valgus. It adds beauty and function to the part in line with the rest of the body. If a fracture occurs about the elbow, this angle can be varied or even made into a cubitus varus at the will of the operator. The bone unites with equal facility whether the parts are placed in a valgus, a neutral or a varus position. Similarly, obtaining a valgus of the hip fracture does not by itself imply union. However, obtaining a valgus at the hip in face of the fracture has much to do with length, symmetry and function of the limb. A well-reduced hip is locked bone to bone in a valgus position, for, in this position, maximum length and function are obtained. Poorly reduced or ill-chosen hips do not unite promptly, whether or not they are placed in a valgus position (see Figs. 4 & 7).

CLOSED REDUCTION OF FEMORAL NECK FRACTURES

Indications

Only transcervical fractures can be reduced and fixed successfully. There is no way to lock and hold a subcapital fracture firmly and routinely to its neck, and these

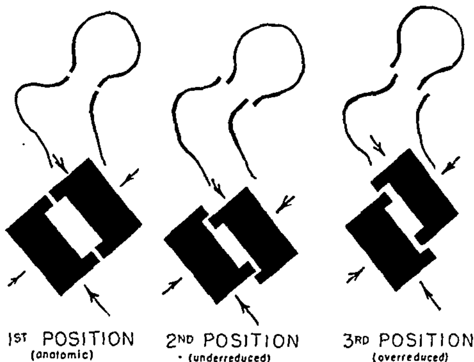
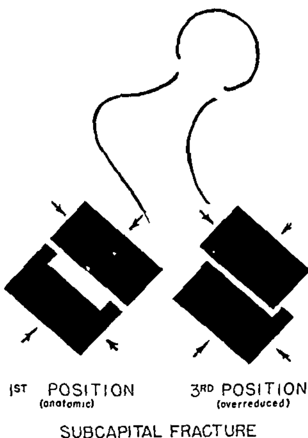


FIG. 20. *(Top)* Showing the mechanical soundness of the principle of the reduction advocated. Represented on the blocks are the medial (inferior) and the lateral (superior) cortical projections of the head and of the neck. Locking can occur only in the "third" position. All other positions are folly. *(Bottom)* Here we see the mechanical setup of a subcapital fracture. Note that no cortical projection is on the head that will allow the gaining of a "third," or favorable, position. Peculiar anatomic circumstances, as shown in Figure 17, occur at times, but in these circumstances an immediate osteotomy is a more dependable way of obtaining union. The only way that the "third" position can be obtained routinely in a subcapital type of fracture is to turn the neck up to prevent the head from sliding away from or falling off its neck. This fact is illustrated by the blocks on the right. Compare with Figures 22 to 25. (McElvenny, R. T.: Quart. Bull. Northwestern Univ. M. School 24:274 & 276)



fractures should not be chosen for closed reduction and pinning. Properly reduced and fixed transcervical fractures unite with bony union rapidly (10 to 12 weeks) almost without exception.

THE RATIONALE

Traction

Traction must be severe. A pull of 80 to 100 pounds on the affected side is essential to achieve actual medial displacement of the femoral shaft. To accomplish this, the following points are important.

A The feet are wrapped from above the ankle to the toes in layers of sheet wadding and then are bound firmly to their supports by multiple turns of muslin bandage, starting well above the ankle. Following this, a complete roll of 2-inch wetproof adhesive tape is used to cover the muslin wrap firmly. The feet must not escape their confines. If they do, all control of the limbs is lost, and failure to achieve reduction follows. During

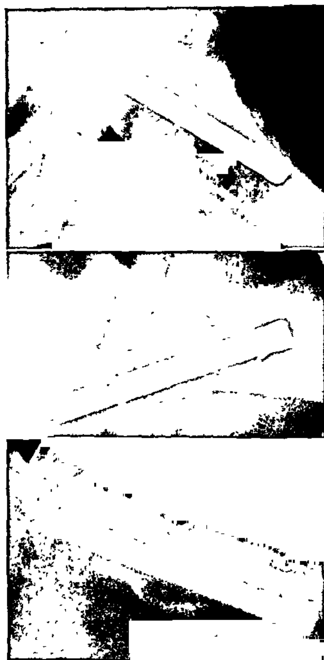


FIG. 21. *(Top)* A true lateral view of an intracapsular hip fracture is as informative diagnostically as an anteroposterior view. Here illustrated is a markedly inadequate (underreduced) intracapsular hip fracture. Note the shortened neck, although the lateral is well posed and shows a true view of the neck Note that the head is posterior (dorsal) to the neck. Note how the head hangs below the neck on the lesser trochanteric side. Note how the neck projects above the head on the side of the greater trochanter. An osteotomy was done on this patient but, due to a technical error, 3 weeks postoperatively it was an absolute failure—the only failure in 18 osteotomies. A lateral view that shows this position or any overlap between neck and head means that the fracture is not reduced. This figure is comparable with that in Figure 9 E.

(Center) This is an anatomic reduction and is the lateral view of Figure 7. Note how head and neck line up. They are anatomic, and this means that reduction is inadequate. This illustration is comparable with Figure 9 D

(Bottom) Showing an adequate reduction; it is comparable with Figure 9 G. This lateral view shows full neck length, and the head on the greater *trochanteric side is above the neck.* There is absolute alignment, as regards a straight line, between head and neck. The internal rotation is complete and slightly eccentric (overreduced) so far as the spicule on the head is concerned To understand hip reduction, one must understand the interpretation of lateral views, and those shown here are deserving of serious contemplation. This view is of the hip in Figure 16.

the operation the feet turn blue, but no complication has occurred.

B. The perineal post must be well padded where it comes into contact with the patient. If a roentgenographic head is slipped between the thighs for lateral views, the normal limb may be abducted, raised or lowered, but traction must be maintained on this limb to keep the pelvis level The perineal post must not be over 2 or 3 inches in diameter. If the post is larger, it may prevent medial displacement of the femoral shaft by pressing against the soft tissue of the thigh when the affected limb is adducted to neutral (see Fig. 5).

C. The pelvis must remain level on the table. This is accomplished by first pulling down on the normal extremity sufficiently to set the pelvis against the severe pull that is later applied to the affected limb. If the pelvis shifts to the affected side and then depresses, a relative abduction is produced. This interferes with true medial displacement of the femoral shaft and prevents true adduction of the limb. A proper reduction is then practically impossible (see Figs. 6 & 15).

D. Traction of the severe type is maintained and never released until the fixation material has been placed correctly and the wound is being closed (see Figs. 7 & 16).

The Hip Capsule and Internal Rotation

The capsule is a tough fibrous envelope and at normal tension fits the head and the neck well. In fracture through the neck with overriding, the capsule becomes relaxed and allows external rotation of the neck on the head, with resulting disengagement and bypassing of the fragments. Under severe traction the capsule is stretched to its limit so that it presses, molds and subtends the head and the neck and furnishes a guide or a template for lining up the fragments. *From the lateral view, anything less than complete internal rotation of the limb fails to get complete reduction of the fracture.* The reason is that, since the posterior capsule is tense, the neck fragment cannot go into internal rotation farther than the point where the neck hits the posterior capsule. Under these circumstances, marked overreduction is practically impossible. If internal rotation is not full, the complete buttress *(calcar femorale)* is not completely under the whole head. This can and does lead to failure of the bony mass on the head to invaginate the neck. *This mass, if small and posterior on the head, will then stay posterior to the neck and fail to contact, and thus lock into, the neck.* Complete internal rotation is gained when the patella points directly medially and the medial condyle of the femur points directly downward (see Figs 8, 9, 11 & 12).

Medial Displacement of the Shaft

This is accomplished with the leg in abduction under severe traction and in internal rotation. The operator places his hands on the great trochanter and pushes firmly downward and medially (see Figs. 14-17).

Adduction of the Limb

After internal rotation and medial displacement have been proved by roentgenograms, the traction is maintained, and the leg is adducted to neutral, with internal rotation also maintained. A roentgenographic check now is taken in both the anteroposterior and the lateral planes. If the medial displacement is still present with the limb in adduction, no angulation force will be exerted against the fixation material because the shaft actually is displaced, and the weight thrust through the shaft is in direct line upward and for the most part falls medial to the head (see Figs. 8, 9 & 14).

Roentgenographic Views

Anteroposterior View. The only view that is of diagnostic significance is one with the limb in complete internal rotation. In this position the full length of the neck is shown. At the time of reduction complete relief of both the head and the neck is obtained for study, due to the traction and the internal rotation. This view determines whether one is dealing with a subcapital or a transcervical fracture. From this view correct or incorrect reduction is determined, and the progress of the fracture can be followed accurately. Any anteroposterior view with less than complete internal rotation of the limb fails to be of diagnostic significance (see Figs. 4, 7, 8, 9 & 17).

Lateral View. A true lateral view is as good a diagnostic aid as an anteroposterior view. Anything less than a true lateral view is an oblique view and is useless for judging a reduction. Complete internal rotation of the shaft must be present to obtain a true lateral view during hip pinning. Frogging the limb has no place in this procedure. Furthermore, under traction, and with the setup advocated here, frogging the limb is impossible.

It is evident that unless a true lateral view shows the fixation material centering the head and the neck, the spherical shape of the head will let the nail come through the head and contact the acetabulum and never show this unfortunate circumstance in the anteroposterior view. Therefore, it is axiomatic that, unless the fixation material

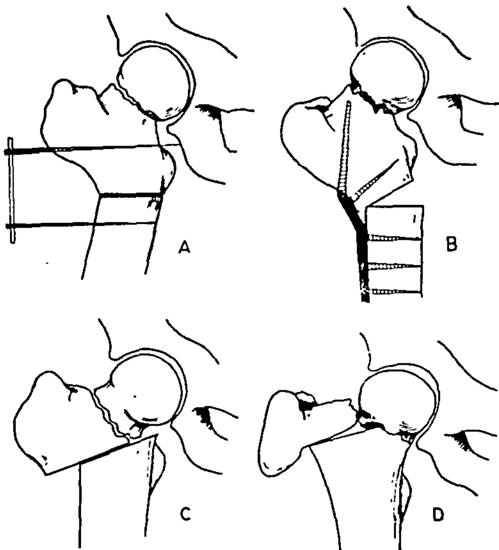


FIG. 22. Various types of osteotomies (after James Dickson). (A) Haas. (B) Blount. (C) McMurray (the Reich addition is an exquisite modification of this conception). We have added severe traction and the Steinmann pin. The traction regains length, throws the head into valgus and obtains contact by physiologic compression, which, to the author's way of thinking, is sufficient for bony union anywhere. The Steinmann pin gets the greater trochanter down the shaft and out for good leverage. The pin also maintains internal rotation and medial displacement of the neck until healing has progressed far enough to maintain the situation (see Fig. 23). (D) Leadbetter. This is a valuable procedure for certain subcapital fractures and certain slipped upper femoral epiphyses. A procedure such as this and the others demonstrated here show that these men may not have advocated *correct* (accepted) procedures but they certainly favored procedures that accomplished the purpose. Between these two conceptions is a canto as big as life itself.

centers the head in the lateral view, the lateral view, not the anteroposterior view, must be used to judge the nail depth (see Figs. 9, 14 & 21).

The Fixation Material

Multiple small pins, threaded only at the point, are excellent in discouraging rotary motion of the femoral head upon its neck. The only disadvantage for many is that the pins are more difficult to insert correctly. For this reason, these pins may diverge so widely that the head does not settle well on the neck, locking may be prevented, and failure results.

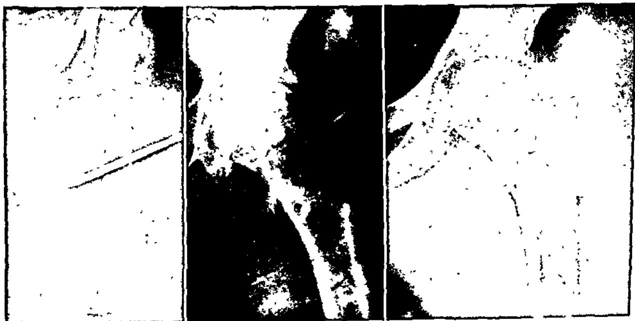
According to reports, lag screws or spring tension screws also serve well if the head is transfixed and drawn down tightly upon the neck to ensure locking of bone to bone. In some cases, the hole in the femoral shaft must be larger than the shaft of the screw. If firm locking of the head and the neck is not achieved, the further settling of the head upon the neck can cause upward tilting of the shaft of the screw, with falling-away of the head and consequent failure. All types without plate have the same disadvantage as the cannulated nail.

The cannulated nail makes an excellent rod for the head to be guided onto the neck.



FIG. 23. *(Top, left)* Shows the original pinning a few weeks after fracture. Note that the nail is well placed but that the reduction is inadequate. It lacks medial displacement. The neck fragment on the head is below and medial to the neck. Failure in this situation is predictable *(Top, center)* Sixteen months later. The nail was thought to be causing the pain in the hip and was removed 2 months previous to the taking of this roentgenogram. Delayed (fibrous) union is evident. *(Top, right)* Under severe traction the head separates from the neck, proving fibrous union. This type of cohesiveness between head and neck prevents the establishment of normal and, therefore, an adequate blood supply. Note that overpull has established length. The rod is anterior to the neck and is skewered in to establish line of osteotomy (Reich).

(Bottom, left) The osteotomy is completed, and displacement and internal rotation are considered to be adequate. Note the rod that is thrust through the greater trochanter, which then is levered down the shaft. Then the rod is drilled through the shaft to maintain the trochanter, internal rotation and medial displacement. It allows free compression forces to settle the head on the neck, although this rod is incorporated into the spica. It is spun out 3 weeks later *(Bottom, center)* In plaster after rod has been spun out. Note the degree of abduction. More abduction than this increases medial displacement and will give overcorrection and marked knock-knee deformity. *(Bottom, right)* Union obtained. The patient is free of plaster, and weight-bearing has been started.



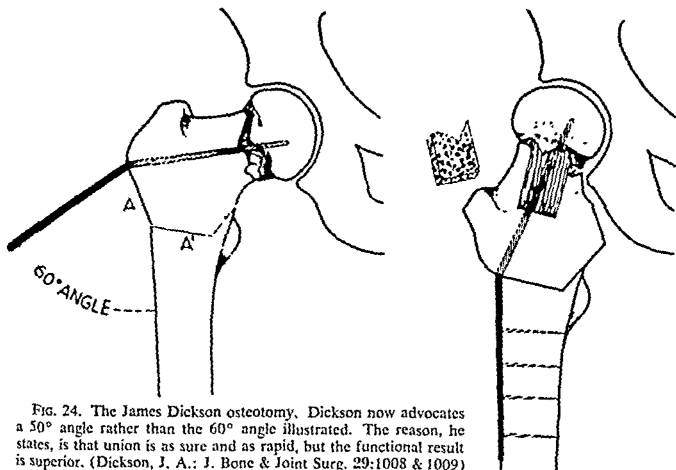


FIG. 24. The James Dickson osteotomy. Dickson now advocates a 50° angle rather than the 60° angle illustrated. The reason, he states, is that union is as sure and as rapid, but the functional result is superior. (Dickson, J. A.: J. Bone & Joint Surg. 29:1008 & 1009)

It is strong and broad and thus prevents external rotation of the neck upon the head. It can be inserted easily and accurately parallel with the *calcar femorale in the lower* half of the head and the neck. If it is placed to a depth of within ¼ inch of the articular surface of the femoral head, its subtending properties are improved. In spite of its size it does not seem to harm the femoral head.

It is a simple matter to drill a ¼-inch hole in the shaft, extending into the neck. The guide wire is run through this hole. By probing with the wire to the medial cortex of the femoral shaft and feeling one's way upward from this, one finds that the wire usually will follow the *calcar femorale* and end low in the neck, parallel with the *calcar femorale,* provided that the drill hole is started far enough below the greater trochanter.

Roentgenograms will show the positioning of the wire. If it is incorrect, the next wire is put alongside the first, *the angles* are varied easily and the depth is altered by studying the roentgenogram. The second wire is put home, and the first wire is removed. Roentgenograms now prove the *position of the wire in both planes. If* placement is satisfactory, depth is adjusted, if necessary, and the nail length is measured. *Then the wire is driven through the head and into the acetabulum—before inserting the nail.* The deep wire prevents the head from spinning as it accepts the nail.

The disadvantage is that the nail does not discourage small rotary motions of the head upon the neck. The nail is only a rod; its purchase in the head is similar to the engagement of a femoral rod in a supracondylar fracture of the femur. This purchase, in both instances, is inadequate. Most fractures of the transcervical type lock bone to bone in such a manner that they eliminate shear, angulation and torque. However, due to peculiar anatomic factors, there are certain fractures that do not change position but *allow some rotary motion.* In spite of invagination this motion is present.

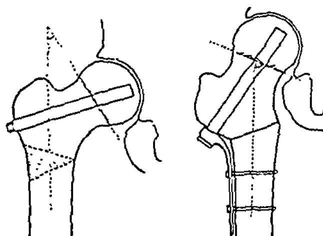


FIG. 25 A. The J. C. McNeur modification of the Dickson osteotomy. (Fig. 25 A-C *in* McNeur, J. C.: J. Bone & Joint Surg. 35B:189 & 190)

LENGTH OF BONE TO BE REMOVED AT THE BASE OF THE WEDGE FOR VARYING BONE DIAMETERS TO PRODUCE A GIVEN ANGLE OF ABDUCTION

DIAMETER OF BONE (INCHES)	ANGLE OF ABDUCTION REQUIRED 20°	30°	40°	50°	60°
	Base of wedge in inches				
1.0	0.35	0.53	0.72	0.93	1.15
1.2	0.42	0.64	0.86	1.02	1.38
1.4	0.49	0.75	1.01	1.30	1.51
1.6	0.56	0.85	1.15	1.49	1.85
1.8	0.63	0.96	1.29	1.68	2.08
2.0	0.70	1.07	1.44	1.86	2.31

FIG. 25 C. McNeur's table that establishes the amount of bone to be removed to obtain the correct angle for a chance of maximum union with the best functional result.

FIG. 25 B An example of the procedure from McNeur's article.

A nail placed correctly and deep in a well-reduced fracture usually suffices. The forces are bone to bone, with the metal holding the position. If the forces are bone to metal to bone, failure ensues. The bone must hold the metal; the metal cannot hold the bone. The head settling on the nail, which is in direct line of the weight thrust, extrudes some nail out of the femoral shaft, but the depth of the nail in the head remains constant. Once settling and locking occur, the head cannot drop off the neck unless external rotation of the neck on the head is allowed. However, if the design of the fracture is such that slight rotary motion of the head on the neck is not prevented by locking of the bony fragments, the nail will be slowly milked out of the head. Once the nail starts moving, the head will not shift until the nail is completely out of it. At this time migration of the nail ceases. Since delayed union is present, external rotation of the neck on the head is possible. If this occurs, disengagement of the fragments follows.

It appears that the use of a pin or two in addition to the nail would have prevented the complicating factor of torque that has plagued us occasionally. Once the nail is set, the guide wire sticking out from the nail gives the line for accurate and quick setting of the additional pins whose length already is known from the nail used.

The Pugh nail is composed of two parts. The first part is a unit of sleeve and plate. The second unit is a nail deeply flanged and tapered at the tip with a smooth, keyed shaft to fit the counterpart of the sleeve and thus prevent rotation of the nail in the sleeve. In addition to the above, a friction ring is provided, which offers enough resistance to sliding to discourage the nail from dropping out of the head, but not enough friction to prevent the head from settling upon the waiting neck.

This gadget is similar to the one developed by Massie. The following advantages seem to be apparent: (1) Settling of the head upon the neck is encouraged by the sleeve and shaft action. (2) Tilting of the nail shaft within the femur is prevented by the sleeve and plate. This should prevent the head from falling toward varus due to ill-fitting between the nail and the femoral shaft. (3) Penetration of the nail into the acetabulum is not likely. This allows deep setting of the nail. (4) Wider application of the unit, as it may be used for intracapsular, intertrochanteric or subtrochanteric fractures.

The disadvantages of this nail are fewer than those of the Smith-Petersen. However, this sliding nail will allow slight rotary motions of the head upon its neck. This will cause delayed union and aseptic necrosis of the femoral head. *Adequate reduction, or osteotomy, is still essential if firm locking of bone to bone is to be achieved.* If the reduction or osteotomy is not proper, the nail will move out of the head or cut through as the head travels its fateful way to varus. Additional pins are still essential to prevent the slight rotary motion that occurs in some fractures in spite of adequate reduction and locking of bone to bone. This locking prevents tipping of the head away from the neck but will not always lock tight enough to prevent the slight motion at the fracture site. For this reason, an extension should be made above the sleeve to allow the quick insertion of pins, threaded on the end, to be run parallel with the nail and into the femoral head to discourage this rotary tendency. This template could be attached by a male counterpart to the sleeve and be used over and over for either the insertion of a pin or a bone graft. Cannulated drills should be furnished to fit over guide pins in case bone grafts are desired.

Further, the design is awkward if one is to combine an osteotomy with hip pinning. The reasons for this are: (1) Once the nail is set into the head, the fitting of the plate to the shaft is awkward following osteotomy and shaft abduction. (2) Bending and molding of the plate take time and put strain and torque forces upon the metal and the proximal bone fragment. (3) The sleeve

is too long for routine use. An ideal design would incorporate the principles of the sliding nail with the McLaughlin adjustable plate. Such a nail is now in production. This new nail should serve in most fracture situations about the hip, including primary osteotomy at the time of pinning.

The Procedure

1. The feet are bound, and the pelvis is set by traction to the normal extremity.

2. The affected limb is rotated externally, and traction is applied. External rotation allows the neck to bypass the head.

3. With the limb in external rotation, it is placed in about 50° of abduction, and more traction is applied. The adductors are tense and stand out. The pelvis remains level.

4. An assistant loosens the rotation screw to the footpiece. As he rotates the foot internally, the operator at the same time rotates the limb at the knee. Full rotation should occur easily and with little effort. If this is not the case, more traction is needed to get the neck under and free from hitting the femoral head.

5. When full rotation is gained, the operator places his hands upon the greater trochanter and pushes down firmly and medially.

6. With traction maintained and increased slightly, the limb is adducted to neutral. Next, roentgenograms are taken. If these are of quality and reduction is achieved, the table arms are set, and the patient is prepared and draped.

7. Pinning is accomplished through a lateral incision. A starter is threaded over the guide wire and macerates the cortex; the nail then has a close fit with the shaft. After the roentgenogram proves the procedure correct, the heavy traction is released, and the wound is closed (see Figs. 14, 17-20).

8 Once the nail has been set, impaction of the fragments is not indicated or essential. Some men first obtain adequate reduction and then release traction. If locking and invagination occur, the guide wire is inserted and the pinning is completed. If, on the other hand, a Massie or a Pugh nail is used, it is first set. Traction is then released and the fragments are impacted without beating the head up. In any of these methods, following completion of the nailing or nailing and impaction, the small torsion pins are inserted (Figs. 15-19).

THE OSTEOTOMY

Indications in the Adult

1. In fresh fractures of the hip this operation is a must in the subcapital type.

2. This operation is indicated in transcervical fractures that cannot be reduced adequately for any reason.

3. At the first sign of delayed union or nonunion an appropriate osteotomy is indicated.

4. For old delayed union, for nonunion and many cases of so-called dead heads, a certain type of osteotomy is indicated.

5. For certain painful hips that result from degenerated heads, or in poor mechanical setups following cup arthroplasty, this procedure in one of its forms offers much to many people.

Osteotomy About the Trochanters

This procedure dates back over 100 years In the early 1900's, Lorenz, Haas and others were using this procedure for many conditions about the hip. McMurray used an oblique osteotomy with medial shift of the shaft to relieve pain from degenerated hips. Later, Reich applied more exactness to this procedure and advocated it for repair of nonunion between head and neck. Leadbetter, just before his death, advocated a higher osteotomy through the femoral neck for treatment of nonunion or as a primary method in treating femoral neck fractures (see Fig. 22).

In using the oblique displacement osteotomy, many failures were encountered because severe traction was not used to overpull and restore length before perform-

ing the osteotomy as high as possible and still be able to get shaft displacement. Severe traction should be diminished only following the displacement of the shaft and the fixation of the Steinmann pin in plaster. After traction is released, the muscles retract, and the shaft snugs up tightly against the head and the greater trochanteric fragments. This gives the right compression forces, and union usually follows.

If osteotomy is done at a level for displacement, with no traction and no internal rotation, the fragments have indifferent contact, the compression forces fail to act, and failure often results.

Proper metal fixation is hard to apply with any degree of confidence. Often the plate cuts out, displaces or holds the 3 fragments apart. These cases do best in plaster spicas.

This writer has done 18 of these osteotomies for nonunion of the femoral head and neck during the past 14 years; overpull and plaster spica were used in all. Sixteen of these cases presented excellent results. Regardless of the condition of the femoral head, whether it was degenerated at time of operation or not, the medial displacement of the shaft and the added support to the femoral head have given these people tremendous endurance, absence of pain and normal activity for everyday living. For this reason, oblique osteotomy might have a valuable place in the treatment of painful hips that sometimes follow cup arthroplasty Because of the technical factors and the plaster spica, this type of osteotomy never has had the acceptance it really deserves (see Fig. 23).

Improvements in Osteotomy for Certain Types of Hip Fracture

Dickson's Osteotomy

During the past 15 years, James Dickson has developed an exact angulation osteotomy that has great potential and carries the following advantages in the treatment of intracapsular hip fracture: (A) Limb length is restored automatically. (B) Hip motion is not compromised. (C) Early mobilization without plaster is possible. (D) The metal fixation is firm and dependable at the broad, closely apposed osteotomy site. (E) The same metal fixation penetrates the femoral head similarly to a hip pin and guides this fragment onto the upturned and waiting neck in correct position. This position changes shear and angulation forces to direct compression forces and thus fulfills the criteria for rapid bony union at the fracture site. (F) In fresh fractures a good reduction can be obtained. The fragments are pinned. The osteotomy then converts the fracture line to the proper plane for bone healing. As a primary procedure it offers much, and its hazards are little greater than in hip pinning or in prosthetic replacement. (G) Rapid bony union, with the femoral head maintained, occurs with surprising regularity. (H) Bone can be added across the fracture site when indicated. Indication is in treating delayed union. Bone is added by curettage of the fracture site and packing with bone chips plus small cancellous grafts from the ilium that contact head and neck deeply and adequately. The pinning and osteotomy are done, and following this the curettage and bone addition are done circumferentially (see Fig. 24).

McNeur Modification of the Dickson Osteotomy

This modification deserves serious consideration. Dr. James Dickson has suggested this as a valuable and needed addition to his fundamental conception.

The cuts are made with a handsaw that does not burn or scar. The upper cut is carried three quarters of the way through the bone. Following this, the lower cut is completed first, and then the superior cut is completed. The lower fragment is abducted until firm and complete contact at the osteotomy site is gained. The extension bar is attached to the nail and the shaft, and the wound is closed.

This procedure is valuable in immediate

treatment of recent subcapital hip fracture. If used for delayed union, the addition of bone as described by Dickson should be considered (see Fig. 25). Recently, Moreira advocated addition of bone to the fracture site and neck cavity as a primary procedure.

SUMMARY

1. Epiphyseal bone appears to be more specific than shaft bone.

2. The separated head of the femur is a living graft of specific epiphyseal bone and must be alive and stay alive if it is to propagate itself and function as a femoral head. Replacement by creeping substitution means that the specific epiphyseal bone is dead. The replacement substance is nonspecific and useless for carrying out the destiny of the former specific tissue.

3. Impacted neck fractures that are of the transcervical type in the locked position are the only ones that routinely heal rapidly with true bony union. These should be fixed. All *other impacted fractures, if of the transcervical* type, must be disengaged by traction and properly reduced and pinned. If of the subcapital type, they must be reduced properly and then submitted to osteotomy.

4. Occasionally, subcapital fractures may be impacted and locked at the time of fracture or reduced at operation to the point of locking. An example of this has been shown. This condition is encountered infrequently. Osteotomy of the type advocated is the method of choice in dealing with this fracture.

5. Nonunion is the result of the following: (A) choosing the wrong type of fracture to pin; (B) improper reduction of the right type of fracture; (C) correctly reducing the right type of fracture and then placing the fixation material incorrectly.

6. Delayed union is the most common cause of aseptic necrosis and failure following hip pinning. This condition is not, as is sometimes thought, a refracture when the head shifts or falls off its neck. True bony union never has existed at the fracture site (see Fig. 23). This condition occasionally follows adequate reduction due to rotary motion of the head upon the neck. If it develops, migration or extrusion of the nail from the head usually occurs without marked tipping of the head fragment, although the lateral view often shows positional change of the fragments. If taken early, osteotomy usually induces rapid bony union at the fracture site and also saves the femoral head.

7. The steps in an adequate reduction of a properly chosen hip fracture are: (A) traction that pulls the fragments apart but maintains a level pelvis; (B) external rotation and abduction of the limb; (C) complete internal rotation of the femoral shaft; (D) medial displacement of the femoral shaft; (E) maintenance of traction, internal rotation and medial displacement as proved by roentgenograms when the limb is adducted to a neutral lateral position; (F) absolute fixation of this position by firm locking of all table attachments; (G) proper placing and proper fit of the fixation material; (H) releasing the severe traction only after the fixation material is placed permanently in proper position and proved by roentgenograms.

8. The object of closed hip reduction is to choose a transcervical type of fracture and manipulate it so that the inferior medial portion of the head fragment drops into the medullary canal of the neck.

9. Certain types of osteotomy are indicated in subcapital types of hip fracture. Osteotomy is indicated at the first sign of nonunion or delayed union.

10. A lateral roentgenogram properly taken is as good a diagnostic aid as an anteroposterior view. In certain instances the lateral is used to judge the depth of the fixation material.

11. In checkup roentgenograms, the anteroposterior should be viewed prior to frogging the limb. If, in adequately reduced hips, delayed union is present and without symptoms, the metal has migrated from the

head, or, in other cases, where a similar situation exists, careless frogging will disengage the fragments. This calls for not only an osteotomy but reduction and repinning as well.

12. Hips that are *going to unite* present (A) adequate reduction, (B) full control of the limb, (C) no pain or limp, (D) no migration of metal, *and* (E) *no condensation* at the fracture line, no shift of fragments and no productive reaction about the fracture site.

13. Most of the poor results following the treatment of intracapsular hip fracture are from poor treatment of intracapsular hip fracture.

CONCLUSION

The contentions brought forth here can easily be proved right or wrong. All that is necessary is to induce others to duplicate the standards and the method mentioned. Sometimes this is difficult, because, in order to induce many to try, it is necessary first to persuade them to lay aside old thoughts, habits and procedures.

Getting immediate bony union of the femoral head to its neck is the only way to improve the present results of intracapsular hip fracture. This article has been written to encourage a more optimistic attitude toward this problem. Whether one routinely reduces a hip adequately and then osteotomizes each case or whether one reduces and pins the transcervical fracture and osteotomizes all others matters little if rapid bony union at the fracture site is obtained uniformly What does matter is that one does not accept nonunion or delayed union as an expected and unfortunate complication of hip fracture. If the correct case is chosen and the right procedure followed, good hips are produced year after year with but few exceptions. The occasional technical error and the ever-present human frailties are then the major factors in precluding perfection, as they seem to be in all cases of continuous human endeavor.

BIBLIOGRAPHY

Dickson, James A.: Biochemics: fractures, femoral neck *in Medical Physics*, vol. 2, p. 94, Chicago, Year Book Pub., 1950.

———: High geometric osteotomy with rotation and bone graft for un-united fractures of the neck of the femur, J. Bone & Joint Surg. 29:1005, 1947.

———: The "unsolved" fracture; a protest against defeatism, J. Bone & Joint Surg. 35:805, 1953.

———: Use of osteotomy and bone grafting in treatment of un-united fractures of neck of femur, J.A.M.A. 137:1199, 1948.

Hass, Julius: The Lorenz osteotomy and its use, Ergebn. d. Chir. 21:457, 1928.

Leadbetter, G. W.: Cervical-axial osteotomy of femur; preliminary report, J. Bone & Joint Surg. 26:713-720, 1944.

Linton, P.: Different types of intracapsular fractures of the femoral neck; a surgical investigation of the origin, treatment, prognosis and complications in 165 cases, Acta chir. scandinav., vol. 90, Supp. 86, pp. 1-122, 1944.

McElvenny, R. T.: An interpretation of certain factors affecting treatment of intracapsular hip fractures, Quart. Bull. Northwestern Univ. M. School 24:267, 1950.

———: Management of intracapsular hip fractures, S. Clin. North America 29:31-58, 1949.

———: Roentgenographic interpretation of what constitutes adequate reduction of femoral neck fractures, Surg., Gynec. & Obst. 80:97-106, 1945.

McMurray, T. P.: Osteo-arthritis of the hip joint, Brit. J. Surg. 22:716, 1934-1935

McNeur, J. C.: Treatment of subcapital fractures of the neck of the femur, J. Bone & Joint Surg 35B:188, 1953.

Moreira, F. E. Godoy, and De Camargo, F. P.: Contribuicão ao tratamento das fraturas do colo do femur, reprint from Rev. Hosp. das Clinicas 12:73-80, 1957.

Pauwels, Friedrich. The fracture of the femoral neck as a mechanical problem; basic conditions of the healing process, prognosis and basic therapy *in* a magazine for orthopaedic surgery published by Ferdinand Enke, of Stuttgart, 1935.

Reich, R. S.: Ununited fracture of the neck of the femur treated by high oblique osteotomy, J. Bone & Joint Surg. 23:141-158, 1941.

Schanz, A.: About walking difficulties after fracture of the neck of the femur, Deutsche med. Wchnschr. 51:730, 1925.

Tucker, E. J.: The preservation of living bone in plasma, Surg., Gynec. & Obst. 96:739, 1953.

Le Tractamento Immediate de Fractura Coxal Intracapsular

Summario in Interlingua

1. Il pare que le epiphyse de ossos es plus specific que le diaphyse.

2. Le separate capite del femore es un graffo vive de specific osso epiphysic. Illo debe esser vive e remaner vive pro propagar se e pro functionar como capite femoral. Su reimplaciamento per substitution progressive signala que le specific osso epiphysic es morte. Le substantia substitutive es nonspecific e noncapace a plenar le functiones del defuncte histos specific.

3. Impactionate fracturas cervical, que es del typo transcervical e que se trova in position claudite, representa le sol casos in lor gruppo que se coalesce routinarimente e rapidemente in un ver union ossee. Iste fracturas require fixation. Omne altere fracturas impactionate, in tanto que illos es del typo transcervical, debe esser reducite per traction con clavage subsequente. Si illos es del typo subcapital, le reduction debe esser sequite per osteotomia.

4. Il occurre sporadicamente que fracturas subcapital es impactionate e claudite al tempore del accidente o es reducite al operation de maniera a attinger clauditura. Un exemplo de iste genere ha essite presentate Le condition non se incontra frequentemente. Osteotomia del typo recommendate in le presente articulo es le methodo de election in tal casos.

5. Non-union resulta (a) del selection injudiciose de casos de fractura pro le manovra del clavage, (b) del reduction incorrecte de casos de fractura que es correctemente selectionate pro iste manovra, e (c) del placiamento incorrecte del material fixatori in casos de fractura que es correctemente selectionate e correctemente reducite.

6. Union tardive es le plus commun causa de necrose aseptic e de non-successo in casos de clavage coxal. Tal non-successos, in que le capite se displacia o glissa ab su cervice, non es—como certe autores opina—le effecto de un fractura. De facto, nulle ver union ossee ha unquam existite al sito del fractura (Fig. 23). Iste condition pote occurrer mesmo in despecto de un reduction adequate. Illo es alora le effecto de un motion rotatori del capite super le cervice. In tal casos, migration o extrusion del clavo ab le capite occurre usualmente sin marcate inclination del fragmento capital, ben que le aspecto lateral exhibi frequentemente alterationes positional del fragmentos. Osteotomia, si interprendite promptemente, induce usualmente un rapide union ossee al sito del fractura e succede etiam a salvar le capite femoral.

7. Le pasos successive in le reduction adequate de un ben selectionate fractura coxal es: (a) Traction que separa le fragmentos sed mantene le pelve in position nivellate. (b) Rotation externe e abduction del extremitate. (c) Complete rotation interne del diaphyse femoral. (d) Displaciamento medial del diaphyse femoral. (e) Mantenentia del traction, del rotation interne, e del displaciamento medial, evidentiate per radios X, quando le extremitate es adducite al position lateral neutre. (f) Fixation absolute de iste position per le firme blocage de omne accessorios del tabula orthopedic. (g) Placiamento e adjustamento correcte del material de fixation (h) Relaxation del traction sever solmente post que le material de fixation es permanantemente placiate in le position correcte e post que iste facto ha essite verificate per radios X.

8. Le objectivo del claudite reduction coxal es le manipulation de un ben selectionate typo transcervical de fractura, de maniera que le portion medial inferior del fragmento capital cade a in le canal medullari del cervice.

9 Certe typos de osteotomia es indicate in typos subcapital de fractura coxal. Osteotomia es indicate si tosto que on nota le prime signo de non-union o de union retardate.

10. Le roentgenogramma lateral, correctemente executate, es tanto diagnostic como le vista antero-posterior. In certe casos le viste lateral es usate pro estimar le profunditate del material de fixation.

11. In roentgenogrammas consecutori, le aspecto antero-posterior debe esser examinate ante le replication del extremitate. Si in casos de adequate reduction coxal, il ha union retardate sin symptomas o migration del metallo ab le capite, o si in altere casos il ha un simile situation, le injudiciose replication del extremitate pote resultar in le separation del fragmentos. Iste situation require alora non solmente osteotomia sed etiam reduction e re-clavage.

12. Fracturas coxal pro que un bon union ossee pote esser expectate satisface le sequente criterios: (1) Le reduction es adequate. (2) Le extremitate es perfectemente sub le controlo del subjecto. (3) Il ha nulle dolor e nulle claudication. (4) Il non ha migration de metallo. E (5) il ha nulle condensation del linea del fractura, nulle displaciamento de fragmentos, e nulle reactiones productive circa le sito del fractura.

13. Le majoritate del inadequate resultatos in le tractamento de fracturas coxal intracapsular es resultatos del inadequate tractamento de fracturas coxal intracapsular.

Conclusion.—Le assertiones hic formulate es facile a provar o disprovar. Le sol condition preliminari esserea inducer alteros a duplicar le standards e le methodos mentionate. Isto non es facile in omne casos, proque a fin de inducer alteros a interprender un tal experimento, on debe primo persuader les a abandonar ancian ideas, habitudes, e technicas.

Effectuar le union immediate inter le substantias ossee del capite femoral e su cervice es le sol maniera possibile de meliorar le resultatos currentemente obtenite in le tractamento de fracturas coxal intracapsular. Iste articulo esseva scribite pro incoragiar un conception plus optimista del problema. Il non importa si on seque le routine de reducer adequatemente le coxas e de usar osteotomia in omne caso individual o si on prefere reducer e clavar fracturas transcervical e usar osteotomia in omne altere casos, providite que un rapide union ossee al sito del fractura es effectuate uniformemente. Lo que importa, del altere latere, es que on refusa acceptar non-union o union retardate como infelice sed inevitabile complicationes del fractura coxal. Si le casos es ben selectionate e si le correcte methodos es observate, bon coxas es producite in longe series con pauc exceptiones. Alora sporadic errores technic e le semper presente insufficientia human es le principal factores que preveni le attingimento del perfection ideal. Sed isto es ver in omne campos de continue effortios human.

25

Treatment of Intertrochanteric Fractures by Skeletal Pinning and External Fixation*

IRVIN H. SCOTT, M.D., F.A.C.S., F.I.C.S.†

Today there is much written into the literature describing different methods and technics for the treatment of intertrochanteric fractures of the hip. Probably all these methods have merits, and many have disadvantages. I would like to discuss the treatment of such fractures by skeletal pinning and external fixation as the method of choice for the following reasons: (1) It is a satisfactory method of treatment because it is applied easily, it provides adequate immobilization, its application produces a minimal amount of shock to the patient, and it allows early ambulation for the patient. (2) I desire to present a brief description of the technic of its application for the benefit of those surgeons who are not acquainted with the principles of the method. (3) I wish to defend skeletal pinning and external fixation as an acceptable method of treatment for this type of fracture and to correct some of the unjust and unfair criticism of which the method has been a victim.

TYPES OF INTERTROCHANTERIC FRACTURES

For all practical purposes, so far as the treatment by this method is concerned, there are only 2 types of intertrochanteric fractures of the hip (Figs. 1 & 2). Figure 1 is a schematic sketch of that more or less simple fracture which involves only the extreme base of the neck of the femur or the trochanter and in which the medial side of the neck (the supporting structure of the hip) is not destroyed by comminution. The distribution of the pins in this fracture need be only in the upper portion of the femur, 2 or 3 pins being inserted through the lateral cortex of the femur below the fracture, transversing the fracture line and extending into the neck and the head of the femur. Two more are placed through both cortices of the femur at angles to one another below the fracture line, and then all pins are connected by external fixation rods as shown.

The second fracture is the type demonstrated in Figure 2. It is a more complicated type of fracture in which the medial side of the neck of the femur is destroyed either by severe comminution of the lesser trochanter and the medial side of the neck, or that type in which the lesser trochanter and the medial side of the neck are a part of the distal fragment. With this type of fracture it is necessary to distribute the pins the full length of the femur to obtain better leverage. Two or 3 ins are placed through the lateral cor-
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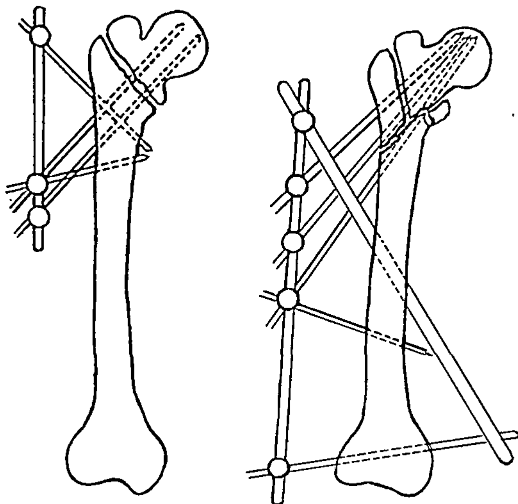
* Presented at the meeting of the American Fracture Association, Miami Beach, Fla., September, 1954.

† Sullivan, I

pins allows the proper leverage to prevent upward and either medial or lateral displacement of the distal fragment.

The proper pin sites are extremely important for obtaining a satisfactory result, and these pin sites are described beautifully by Dr. H. D. Junkin.* Figure 3 is copied from Dr. Junkin's work, which is very important to any surgeon regardless of his standing or ability should he attempt to apply this technic in the treatment of intertrochanteric fractures or any other type fracture. Except to say that in Figure 3 pins 3 and 4 are the ones which transverse the fracture and extend into the neck and the head of the femur and that pins 1 and 2 are the pins which form the distal unit, there

* Junkin, H. D.: The topography of pins; precision pinning of fractures, Indust. Med. 13:887-897, 1944.



Figs. 1 and 2. The two types of intertrochanteric fractures showing the distribution of pins for each type

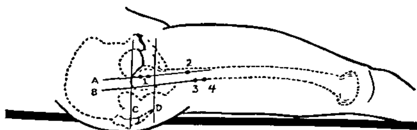


Fig. 3. Topography of pins for intertrochanteric fractures. (Junkin, H. D : Indust Med. 13:887)



FIG 4 Anteroposterior roentgenogram of properly placed pins.



FIG. 5. *(Top)* Interval and *(bottom)* completed dressings.

is no need here for a detailed description of the topography of pins. (Reprints of Dr. Junkin's work are available.) Figure 4 is an anteroposterior roentgenogram showing the pins properly placed

A very simple and extremely important step in the use of this technic, as absurd as it may sound, is the application of the proper dressing. Following the proper insertion of the pins and the application of the external fixation rods, fluff gauze in large quantities, possibly supplemented by sterile mechanics waste, is packed tightly around the pins and between the fixation rods and the skin to compress and immobilize the soft tissue. Then an elastic bandage is wrapped around the large dressing, and over this adhesive tape is applied to seal the dressing completely. This is a somewhat time-consuming procedure, particularly when the operator is already tired, but it will save much time for changing dressings later. If this dressing is applied properly initially, there will be very little, if any, drainage around the pins because the soft tissue will have been immobilized, and the motion of the soft tissue on the pins is the main etiologic factor of pin drainage. Figure 5 shows a partially applied dressing and the same dressing after completion.

The most common criticisms of skeletal pinning and external fixation of hip fractures are profuse drainage around the pins, osteomyelitis from the use of pins and pain at the pin sites In almost every instance these complications are due to faulty technic. Drainage around the pins always is caused initially by pressure on the soft tissue against the pins, giving rise to necrosis of that tissue involved. If the pin is inserted in a direct line from the point of entrance into the soft tissue to the point of entrance

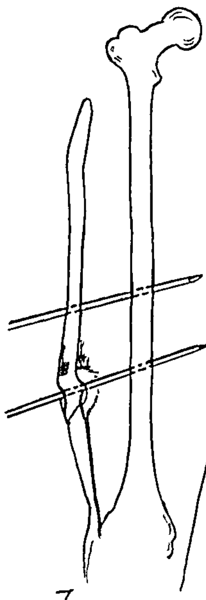


FIG 6 Lower pin inserted improperly causing soft-tissue necrosis. Upper pin is inserted properly showing pin in a direct line from the point of insertion in the skin to the point of insertion in the bone



FIG 7 *(Top)* Clean pins after removal of initial dressing in 12 weeks. *(Center)* No infection round pins. *(Bottom)* Small pin holes immediately following removal of pins in 12 weeks.

into the bone, there will be no pressure on the soft tissue and, consequently, no pressure necrosis with resultant drainage. Occasionally, when a pin is inserted at an angle closely paralleling the bone, there is difficulty in engaging the pin in the bone because it slips. In this event, after the pin has been inserted in a direct line through the skin to the point of entrance in the bone, the pin can be brought upward until it is more nearly perpendicular to the bone and drilled until it is engaged; then the pin is brought back to the initial and final position, releasing the tension on the soft tissue. Then the insertion is completed. In Figure 6 the top pin shows proper pin insertion, and the lower pin shows incorrect insertion with resultant soft tissue necrosis and drainage. If the pins are inserted as demonstrated by the top pin in Figure 6, and a dressing applied,

there will be only a very slight serous drainage which will form a crust or a scab around the pin sealing off the only point of entrance to any infection. The initial dressing, if properly applied, should not be changed, because in doing this the crust or the scab usually is removed with the dressing, leaving a small raw surface which gives rise to more drainage. Frequent changing of properly applied dressings is absolutely contraindicated. Figure 7 shows the equipment, the pins after the rods have been removed and the pin sites after the pins have been removed in a case in which the initial dressing had been removed in 12 weeks. Obviously, there is no drainage or infection.

After reducing and pinning a hip fracture and all the above precautions have been taken, if there is any evidence of pressure on the skin against the pin it should be corrected by making an incision with a sharp-pointed knife along the pin through all of the soft tissue all the way to the bone until the pressure has been released. Then a dressing should be placed tightly around the pin and left there. Pain around a pin also is due to pressure on the soft tissue.

Osteomyelitis can be caused only by 2 things: (1) unsterile technic and (2) by drilling a pin too rapidly with an electric or some other high-speed drill, causing a thermal necrosis with a resultant ring sequestrum. I have had this happen in two of my cases and I blame myself and not the method of treatment of the fracture.

As is shown in Table 1, we have treated 112 intertrochanteric hip fractures from 1949 to 1953. All cases were followed for a period of at least 10 months (several months after union) and many for a much longer period. The average age of the patient treated was 74 years; the youngest treated was 4 years old and the oldest was 97 years old. There were 33 deaths in this group, giving an over-all mortality rate of 30.3 per cent. The mortality rate means very little to me because in the first 56 cases treated the mortality rate was 15.5 per cent, and in the last 56 cases the mortality rate was 40.3 per cent. This was due to the facts that the last 56 treated were of an older average age group, 3 died of associated injuries, and 2 died of malignant disease. It is needless to state the causes of death because they are shown in Table 1. Senility was listed as a cause of death in those patients who were unable to walk prior to obtaining the fracture and could not get out of bed after the fracture was treated. Several of these patients had arteriosclerosis and had had a cerebral accident months before they had the fracture. It is interesting to note that the average age of those who died was 82.2 years, which is 8.2 years older than the average of those who were treated.

The average number of days the fractures were immobilized with pins was 98 6. This average period of immobilization was increased greatly by some of the badly comminuted fractures. The more simple fractures were immobilized from 7 to 10 weeks The average number of days of hospitaliza-

TABLE 1. STATISTICAL CHART OF 112 CONSECUTIVE CASES OF INTERTROCHANTERIC HIP FRACTURES

Total number of cases from 1949 to 1953	112
Average age of those treated	74
Youngest treated	4
Oldest treated	97
Number of deaths	33
Mortality rate	30.3%
Causes of death:	
1. Coronary occlusion	5
2. Senility (arteriosclerosis, etc.)	18
3. Uremia	2
4. Cerebral hemorrhage	3
5. Sarcoma with lung metastases	1
6. Generalized carcinomatosis	1
7. Death from associated injuries (skull fractures, etc.)	3
Average age of those who died	82.2%
Average number of days immobilized with pins	98.6%
Average number of days in hospital	44.5%
Number of nonunions	0
Number of infections (due to faulty technic)	2

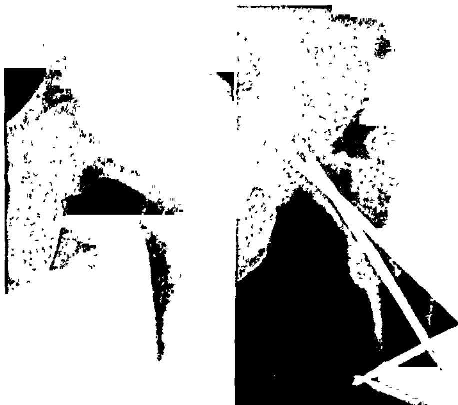


FIG. 8. *(Left)* Initial roentgenogram of left hip. *(Right)* Anteroposterior view after pin fixation.

tion was 44.5, which was lengthened greatly by old people who had no facilities for care at home and were forced to stay in the hospital until they could walk. The younger patients who were physically able to walk on crutches were hospitalized from 10 to 14 days.

There were no cases of nonunion in this group among those who survived There were 2 infections both of which were due to technical error, namely, by drilling a pin too rapidly and producing a thermal necrosis. Fortunately, in both instances these infections were around pins away from the fracture site, and both healed immediately following drainage and removal of the ring sequestrum.

CASE REPORTS

Case I. Mrs. S. B., aged 73, was admitted on June 5, 1951, with the first type of intertrochanteric fracture of the left hip caused by a fall (Figs. 8 & 9). The fracture was reduced and pinned the day of the injury under local procaine anesthesia. She was allowed to be in a wheel chair the second postoperative day and was hospitalized for 43 days, being dismissed July 18, 1951. The pins and the equipment were removed from her leg on August 14, 1951 (64 days). The result was excellent, as is shown in Figure 9. Since treating her for this fracture, I have treated her for a similar fracture of the right hip with the same result.

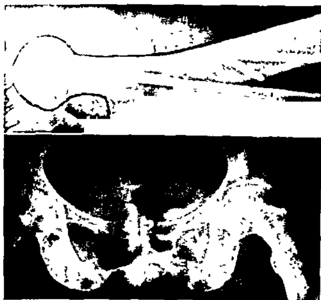


FIG 9. *(Top)* Lateral roentgenogram with pin fixation. *(Bottom)* Fixation removed after 64 days showing good results in left hip. Later the right hip was treated for the same type of fracture.

Case 2. Mrs. C. S., aged 76 years, was admitted to the hospital May 9, 1949, after falling from a moving automobile and sustaining the second type of intertrochanteric fracture of the left hip (Fig. 10). The medial side of the femoral neck was destroyed, and the lesser trochanter was a part of the lower fragment. The fracture was reduced on a fracture table and pinned under ether anesthesia, the pins being distributed from the head and the neck to the condyles of the femur, as is shown in Figure 10.

She was allowed to be in a wheel chair on the third postoperative day and was walking in a walker in 2 weeks. She obtained a satisfactory union, and the pins and the external fixation equipment were removed July 16, 1949 (68 days). Later she developed osteomyelitis in the midfemur, which was caused by my drilling a pin too rapidly, causing a thermal necrosis and a resultant ring sequestrum. On August 5, 1949, she was operated on for the osteomyelitis, the abscess was drained, and the ring sequestrum was removed. Healing took place rather rapidly. On August 19, 1949, while still in the hospital, she was operated on for a right strangulated femoral hernia and made an uneventful recovery from this. She remained in the hospital for 5 months because there were no facilities at home for her care. In spite of all her difficulties, when she was dismissed October 16, 1949, she had full weight-bearing and 100 per cent function of her leg.

Case 3. Mrs. Q. P., aged 49, fell and ob- [illegible]

and external fixation under spinal anesthesia. She was crutch-ambulatory on the 10th postoperative day and was dismissed from the hospital the 27th postoperative day, at which time she returned to her job as a telegraph clerk. The pins were removed March 24, 1950 (76 days). In 4 months she had a 100 per cent functional result.

Case 4. Mrs. V. McC., aged 44, fell from her porch and obtained a very badly comminuted fracture of the left hip on July 5, 1952 (Fig. 12). The fracture was reduced on a fracture table and also by manipulation of some of the fragments with pins. The pins were distributed the full length of the femur and connected with fixation rods She was dismissed from the hospital on July 20, 1952 (15 days), at which time she could walk well on crutches. The pins were removed on November 4, 1952 (121 days), by which time she had a good bone union. She has a 100 per cent functioning leg and walks without a limp.



FIG 10 *(Left)* Intertrochanteric fracture of right hip of the second type described *(Center)* Pins were distributed the full length of the femur Pins were removed after 68 days *(Right)* Final result. Osteomyelitis developed round the pin in the center of the femur from too rapid drilling of this pin.



FIG. 11. *(Left)* Intertrochanteric fracture of the first type. *(Center)* Pins in place after reduction. Pins were removed after 76 days. *(Right)* Good union and excellent functional result in 4 months.

The results of the 79 living cases are shown in Table 2. The classification of results is that of the group at the Massachusetts General Hospital. The results are graduated from Class 0 to Class 4, which is 87½ to 100 per cent. This graduate scale is



FIG. 12. *(Left)* Comminuted intertrochanteric fracture with destruction of the medial side of the neck. *(Center)* Pins and external fixation distributed the full length of the femur. Pins and external fixation removed after 121 days *(Right)* Good anatomic and functional result

Table 2. Anatomic, Functional and Economic Results of 112 Intertrochanteric Hip Fractures by the Classification of the Massachusetts General Hospital

Anatomic	0 - 0 case	
	1 - 3 cases	
	3 - 5 cases	
	4 - 66 cases	83.5%
Functional	0 - 1 case	
	1 - 3 cases	
	2 - 5 cases	
	3 - 3 cases	
	4 - 67 cases	84.8%
Economic	0 - 1 case	
	1 - 1 case	
	2 - 3 cases	
	3 - 3 cases	
	4 - 68 cases	86.0%

Class 0 = 0% to 12½%
Class 1 = 12½% to 37½%
Class 2 = 37½% to 62½%
Class 3 = 62½% to 87½%
Class 4 = 87½% to 100%

then figured on anatomic, functional and economic bases. It is interesting to note that anatomically 83.5 per cent, functionally 84.8 per cent and economically 86 per cent were in Class 4.

CONCLUSIONS

1. Intertrochanteric fractures can be treated very satisfactorily by skeletal pinning and external fixation, provided that the operator understands the principles and the technics of application. Under these conditions, the results compare favorably with those obtained by other methods.

2. Skeletal pinning and external fixation of a hip fracture do not require an open operation and can be achieved quickly with a minimal amount of shock by any general surgeon of average manual dexterity and mechanical judgment.

3. Most criticisms of the method are unjustified and are propagated by surgeons who have tried the method without a knowledge of the technic or condemn it on the basis of hearsay and without any personal experience with the method.

4. In general, the high mortality rate from intertrochanteric fractures is due to old age and not to the fracture itself.

Le Tractamento de Fracturas Intertrochanteric per Medio de Clavage Skeletal e Fixation Externe

Summario in Interlingua

Cento dece-duo consecutive fracturas coxal intertrochanteric es presentate. Illos esseva tractate inter 1949 e 1953 per clavage skeletal e fixation externe. Si le fractura es simple, un sol clavo es placiate al extremitate superior del femore, sed si le fractura es severmente comminutive, clavos es distribuite al longo del femore integre.

Un tense bandage super un grande area circa le clavos es essential pro immobilisar le histos molle in contacto con le clavos e pro prevenir drainage. Le clavos debe esser placiate in un linea directe ab le puncto de insertion al superficie del pelleverso le puncto de insertion in le osso. Isto preveni necrose pressori del histos molle circa le clavo e le subsequente occurrentia de drainage. Le foration del clavos debe esser executate lentemente pro prevenir necrose thermic. Si iste tres principios es observate in conjunction con le usual technica chirurgic, infection e drainage ab clavage skeletal con fixation externe va esser negligibile.

Es demonstrate statisticamente que le causa de morte in fracturas coxal intertrochanteric es usualmente le etate avantiate o un associate vulnere o morbo del patiente e non primarimente le fractura mesme.

Fracturas intertrochanteric pote esser tractate multo rapidemente per clavage skeletal con fixation externe. In multe casos isto es possibile sub anesthesia local con minimal grados de choc in comparation con altere methodos. Usualmente iste patientes deveni ambulatori a un tempore precoce, providite que illes es physicamente valide.

Quatro casos representative es presentate.

26

Turnbuckle Correction of Angulation Deformities of Recent Fractures of the Long Bones*

J. E. M. THOMSON, M.D., AND SCHUYLER P. BROWN, M.D.

Described here is a simple and effective technic for the correction of angulation deformities of recent fractures of the long bones. It has been used in 15 cases, including 2 femurs, 12 tibias and 1 case involving fractures of both bones of the forearm. The technic has been strikingly effective in 13 of the 15 cases but has failed in 2. The 2 failures were predicted even before attempts at correction were undertaken because of the long time which had elapsed following reduction. One of the failures was a fracture of the distal tibia in good approximation, but with an undesirable angulation. Eleven weeks had elapsed following reduction and application of a cast. The other failure was a fracture of the distal femur, also with good approximation but with excessive angulation, and of 13 weeks duration. In both cases, attempts at turnbuckle correction caused considerable pain, persisting as long as the correcting force was applied. Check roentgenograms revealed no diminution in the degree of angulation, and further attempts at turnbuckle correction were abandoned. It was assumed at the time that these patients probably had achieved too firm a union for the utilization of this correction technic. However, in retrospect, it is felt that instead of attempting full correction within an hour or less, the procedure should have been spread out over a week or 10 days, perhaps even longer.

In the 13 successful cases, failure was predicted in 3 because of apparent progress in healing. However, the predictions were erroneous, and the technic was successful. In all 15 cases, the initial reductions were not performed by the authors of this paper or their associates.

In the 13 successful cases, the fractures had been reduced for periods of from 4 days to 3 months. As a guide to the likelihood of success, certain criteria are of importance: (1) the interval which has elapsed following reduction, (2) the character of the approximation of the fragments, (3) the distance of the fracture from the end of the bone, and (4) the roentgenographic appearance of the fracture.

The technic may be utilized in all cases where wedging would be of value, as well as in those cases where union is too firm for the utilization of the ordinary wedging technic but which may be susceptible to the greater and more gradually applied force produced by the turnbuckle. Further, there are advantages to be had in the turnbuckle technic not found in the ordinary wedging procedure. There is little or no likelihood of complete and possibly calamitous disruption at the fracture site, which is known to occur in fresh fractures where the cast has been divided inadvertently and excessive angulation allowed to take place. Also, the

* From the Lincoln Orthopaedic and Rehabilitation Center and the Veterans Administration Hospital, Lincoln, Neb.



FIG. 1. Roentgenograms of fractures of the tibia and the fibula of 3 months' duration prior to correction of angulation.

extent of correction is under the complete control of the operator at all times. If he overcorrects he has only to turn back the turnbuckle an appropriate distance.

In considering the possible disadvantages of this technic, it may be felt that since the hinge affixed to the cast constitutes a fulcrum with the fracture site radially distal thereto, rotation about this fulcrum will cause distraction at the fracture site. Actually, however, the cast and the bone do not constitute one unit There is slippage between the plaster and the padding, between the padding and the skin, and between the skin, the subcutaneous tissue and the bone. In all 13 cases where correction of the angulation was possible, there was no observable distraction in the roentgenograms, and all proceeded to firm union. While contusion of the skin at the point of angulation would seem to be a definite possibility, it did not occur in any case, and it is felt that it never should occur if proper precautions are taken.

In describing the turnbuckle technic,

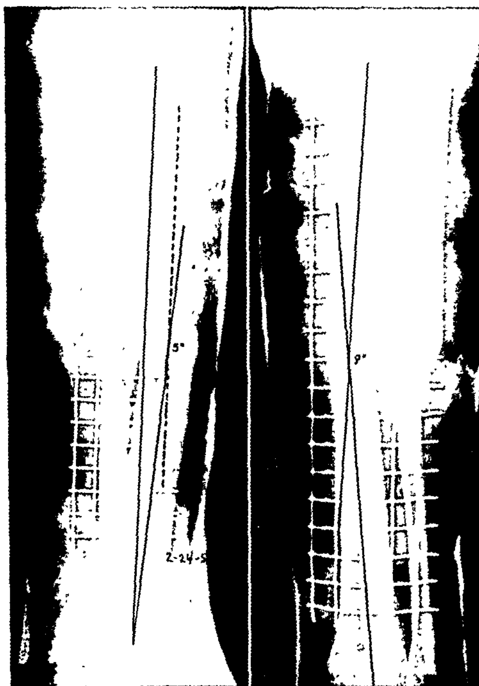


FIG. 2. Same fractures as shown in Figure 1 but with a new cast applied with grid of ½-inch mesh hardware cloth taped to cast.

angulation at the fracture site will be thought of in terms of the deviation of the distal fragment with respect to the proximal fragment, not in terms of the direction of the convexity of the consequent bowing. Thus, in a lateral deviation of the tibia at the fracture site the bowing would be medial.

As it is not always easy to relate precisely the location of the fracture site to the exterior of the cast, anteroposterior and lateral roentgenograms should be taken with the tube centered over the fracture site and a piece of ½-inch mesh hardware cloth taped to the cast with its position marked in pencil on the cast. The grid produced by the hardware cloth on the roentgenogram locates accurately the fracture site with respect to the exterior of the cast.

Figure 1 shows a fracture of the tibia and the fibula of 3 months' duration in only fair approximation with a projection of the lateral deviation in the *anteroposterior* view of 10° and a projection of the posterior angulation in the lateral view of 5°. The cast was bulky, and Steinmann pins were present above and below the fracture site. The pins



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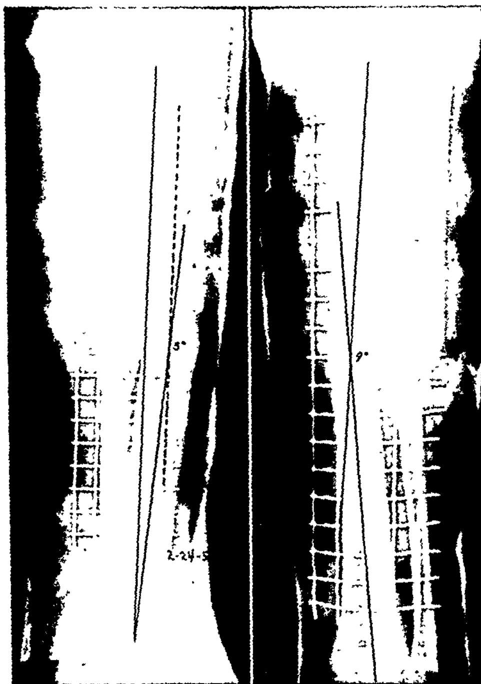


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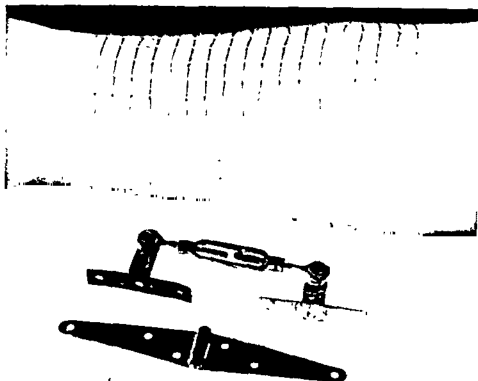


FIG. 3. Lower extremity cast with grid in place, together with equipment used to correct angulation at fracture site.

and the cast were removed, and a new cast was applied. In Figure 2, anteroposterior and lateral roentgenograms of the leg in the new cast are shown with a piece of ½-inch mesh hardware cloth taped to the cast Thus the fracture site is located accurately with respect to the exterior of the cast. In the anteroposterior view, the projection of the lateral angulation is only 9°, while in the former roentgenogram it was 10°. Discrepancies of this magnitude are not infrequently encountered and can be accounted for by slight differences in the positioning of the leg at the times the roentgenograms are taken. It has been found advantageous for the orthopaedist either to position the pa-



FIG 4. Lower extremity cast with hinge and turnbuckle in place and turnbuckle slightly expanded.



FIG. 5. Roentgenograms of case shown in Figures 1 and 2 taken during progress of straightening process.

tient himself or to impress upon the x-ray technician the importance of true anteroposterior and lateral views.

With the direction of angulation of the distal fragment projected on the anteroposterior film as a lateral angulation of approximately 10° and on the lateral film as a posterior angulation of 5°, it is apparent that the true direction of angulation is somewhere between its lateral and posterior projections, but more lateral than it is posterior. Thus, the direction of the correcting force to be produced by the turnbuckle should be in the plane of the true angulation at the fracture site; this is determined with a relatively high degree of accuracy by relating the 10° lateral projection and the 5° posterior projection to the 90° contained in the posterolateral quadrant in which such angulation lies. Thus, in this case, the correcting force was applied in a plan 30° from the frontal plane of the leg. In the cases contained in this series, the direction of application of force in each individual case was based on the operator's judgment rather than precise measurements.

Figure 3 is a photograph of a lower extremity cast with the grid in place, together with the equipment used to correct the angulation. This consists of a 4-inch strap hinge, a 4-inch turnbuckle, two 3-inch "T"-shaped mending plates with the straight arm bent at a right angle to the cross arm and two ¼-inch diameter stove bolts attaching the turnbuckle to the mending plates.

Following the location of the fracture site



FIG. 6. Roentgenograms of fracture shown in Figures 1 and 2 ten days following corrective procedure.

with respect to the exterior of the cast, the cast is cut transversely opposite the direction of angulation, and the strap hinge is affixed with several turns of 3-inch or 4-inch plaster bandage. Then, opposite the hinge, the two "T" plates, with turnbuckle attached, are affixed with several turns of plaster bandage. After 20 or 30 minutes has been allowed for drying, the cast is divided throughout the remainder of its circumference, and the expansion of the turnbuckle is begun.

Figure 4 shows the divided cast with the hinge and the turnbuckle in place and the turnbuckle slightly expanded. The expansion should proceed one turn or less at a time. If the screws of the turnbuckle contain the customary 20 threads to the inch, one complete turn will expand the turnbuckle 1/20 of an inch. If the fracture is very recent, the patient experiences no discomfort. If less recent, some discomfort may be felt, which generally disappears in a few minutes. Another turn, or part of a turn, is then made, and the procedure is repeated until it is estimated that slightly less than the full correction has been accomplished. Then a check roentgenogram is taken. Our experience indicates that if the check roentgenogram is taken shortly following the expansion of the turnbuckle, the ideal correction at that stage is something short of full correction This is because of the fact that the persistence of the pressure produced by the angulation of the cast will further correct the angulation of the bone over the next few



FIG. 7. Roentgenograms of case of undesirable anterior and lateral angulations prior to correction.

days. Hence, if the check roentgenogram shows an overcorrection, or even full correction, the turnbuckle should be turned back an appropriate amount. In cases where the check roentgenograms taken immediately following the expansion of the turnbuckle indicated about 2° less than full correction, roentgenograms taken from 10 days to 2 weeks later showed that full correction had taken place.

Figure 5 contains 3 roentgenograms of the case shown in Figures 1 and 2, taken during progress of the straightening process. The original lateral angulation of approximately 10° has dropped progessively to 6°, 3° and 2°. At the last point, no further expansion of the turnbuckle was made. The wedge-shaped hiatus was filled in with plaster of Paris, and the hinge and the turnbuckle were removed.

Figure 6 shows anteroposterior and lateral roentegenograms of the tibia taken 10 days following the corrective procedure. In the anteroposterior view, the lateral angulation is now down to 0° from the original 10°, and in the lateral view the posterior angulation is now but 2° from the original 5°. In this case attention was directed principally to the lateral component of the angulation, as the posterior component was not functionally significant because of the hingelike character of the ankle joint.

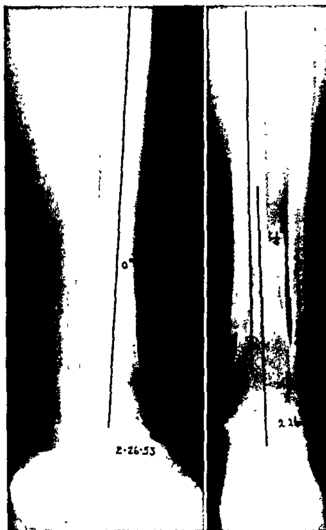


FIG. 8. Roentgenograms of case shown in Figure 7 following correction.

Figures 7 and 8 are roentgenograms of the case in the series in which the shortest period of time elapsed between reduction and correction of angulation—only 4 days. Here there was an anterior angulation of 6° and a lateral angulation of 2°. While the 6° anterior angulation was not regarded as being of great functional significance, the patient happened to be a fastidious woman who would have been highly displeased with any visible deformity of her ankle. In fact,

it was that consideration which caused her physician, who had reduced the fracture, to seek *orthopaedic assistance. Figure 8 shows* roentgenograms taken 9 weeks later with correction of both the anterior and the lateral *angulation.*

Correction de Deformitates Angular de Recente Fracturas de Ossos Longe, Effectuate per Medio de un Lanterna de Tension

Summario in Interlingua

Iste articulo describe un simple e efficace technica pro le correction de deformitates de angulation in recente fracturas de ossos longe. Illo ha essite usate con bon successo in fracturas del femore, del tibia, e del ossos infero-bracial, le quales habeva previemente essite reducite con le resultato de un non-desirabile angulation. In le casos reportate, le correction esseva effectuate inter quatro dies e tres menses post le reduction original.

Le technica pote esser empleate in omne casos in que le cuneation del apparato de gypso esserea de valor e etiam in certe casos in que le union es troppo firme pro le usual technica de cuneation sed in que successo remane possibile per le application del plus grande e plus gradual fortia de un lanterna de tension. Le technica permitte al operante le continue e complete regulation del grado de correction e es exempte del possibilitate de un accidental e forsan calamitose disruption al sito del fractura.

Le technica consiste del sequente mesuras: (1) Roentgenogrammas antero-posterior e lateral a transverso le apparato de gypso pro determinar le exacte sito del fractura e le direction e le grado del misangulation. (2) Le division partial del apparato de gypso al nivello del fractura, opposite al puncto del curvation maximal, e le application, super le division del apparto, de un cardine a alas longe. (3) Le application del lanterna de tension al latere opposite al cardine, sequite per le complete division del apparato. (4) Expansion del lanterna usque al puncto ubi le angulation es correcte.

Section IV

ITEMS

27

The Problem of Handicapped Children in India (Not Including the Blind, the Deaf and the Mute)*

(Spectator Letter)

M. V. Sant, M.D.†

The problem of physically handicapped children is essentially different in the East from that in the West. While physical handicaps in the West are due mostly to industrialization, quick transport and war, in the East they are a legacy of disease, poverty and ignorance.

It is difficult to talk about the problem of the physically handicapped in India. Discussion on such a problem normally should begin with the figures regarding the extent of the problem in terms of the total number of handicapped children in the population. On this point there is no information. The only reliable source for population data in recent years is the census report of 1951, which does not classify the physically handicapped persons in an independent category. Citing certain estimates recently made in the U.S.A., which put the figure of the handicapped population at 20 per 1,000, the Honourable Rajkumari Amrit Kaur, Minister of Health, Government of India, said that the handicapped population in India might be in the same ratio, if not greater, in which case we must have over 7,000,000 handicapped persons in this country. Thus the magnitude of this problem can very well be imagined.

The only other indication of the extent of this problem is perhaps the number of handicapped children that attend the public hospitals. The following observations are based on the figures obtained from the Orthopaedic Department of Jerbai Wadia Hospital for Children in Bombay. This hospital, though located in the city of Bombay, actually draws patients from all over India. Therefore, the conclusions drawn from this small study may be considered as representing the problem of the whole country.

During a period of 10 years (1939-1949), 1,670 children were treated as inpatients in the orthopaedic department of this hospital This does not include those children who were treated as outpatients or those whose parents declined to submit them for treatment, operative or otherwise, for various reasons. The table on page 346 gives the frequency of various conditions for which these children were treated.

From this table it will be seen that today our main problems are tuberculosis and mal-

* The members of the Editorial Board of *Clinical Orthopaedics* think that this Spectator Letter, released November 7, 1952, will be of special interest to our readers Some of this material appeared previously in *The Indian Journal of Child Health*

† Bombay, India.

nutrition. Fortunately, so far, poliomyelitis has never been a serious problem with us, as it has been in the West. It is the impression of most orthopaedic surgeons that in the East we have a comparatively smaller percentage of congenital deformities than in the West. Perhaps this is due to the fact that the ways of life in India are still nearer to nature than those in the West. This is only an impression, and there is no definite proof for this statement.

ORTHOPAEDIC CONDITIONS AT THE B. J. WADIA HOSPITAL*

Condition	%
Tuberculosis of bones and joints	34.1%
Accidental injuries	32.2%
Deformities due to rickets	12.0%
Congenital deformities	11.0%
Deformities due to poliomyelitis	7.6%
Osteomyelitis and suppurative arthritis	2.2%
Miscellaneous conditions	0.9%

These figures compare favorably with those from:

(1) The National Orthopaedic Hospital, Manila, Philippines
(2) The Children's Orthopaedic Hospital, Bombay

The National Orthopaedic Hospital, Manila, Philippines (1950):

Condition	%
Traumatic cases	53%
Postpoliomyelitis	15%
Tuberculosis of bones and joints	14%
Congenital malformations	6%
Osteomyelitis	5%
Others	7%

(Report of the United Nations Conference of Experts on Physically Handicapped Children for Countries of South East Asia, 1950, p. 26)

The Children's Orthopaedic Hospital, Bombay (Total number of cases 978; orthopaedic 889):

Percentages worked out by the author

Condition	%
Poliomyelitis	53.5%
Spastics (cerebral palsy)	24.5%
Tuberculosis of bones and joints	3.1%
Infections of bones and joints	1.7%
Rickets	9.5%
Congenital deformities	5.4%
Postural deformities	0.3%
Traumatic	1.7%

(Fourth Annual Report of the Society for the Rehabilitation of Crippled Children, Bombay, 1951)

* The figures of causative factors at this institution differ slightly, obviously because different types of patients naturally are attracted by the particular type of facilities for treatment of particular conditions.

Presuming that the conditions cited in the tables are responsible for the crippling of children in India, the question naturally arises as to why these deformities cannot be prevented. Developments in the medical sciences have made such strides in the past few years that it should be possible to prevent crippling in most of these children, and in the case of those children where it cannot be prevented, rehabilitation to normal life should not be difficult. In the West, the problem of a handicapped child is one of physical reconstruction. In the East, the socioeconomic structure of our country is the most important factor to be reckoned with. In the West, human values are reigning at a high premium, while in the East, with its vast population, underdeveloped resources, ignorance and lack of education, human life is at a discount, and an attitude of determinism prevents any efforts being made to understand, treat and rehabilitate the physically handicapped. On the basis of experience gained during several years of work in public hospitals, the author attributes the inability of the people to seek treatment for handicapped children to the following reasons: (1) oriental philosopy of life; (2) conditions in public hospitals; (3) fear of surgical treatment; (4) economic considerations; (5) distance from home; (6) ignorance of facilities for treatment.

ORIENTAL PHILOSOPHY OF LIFE

In the East, a handicap in a child is likely to be looked upon as a manifestation of the Will of God and a destiny which cannot be altered. Quite a number of persons would accept it as such and resign themselves to fate. Perhaps this is the best attitude to adopt if the handicap cannot be put right, because, in that case, the child does not develop the psychological complexes so often seen in the West. If the handicap does

not interfere much with the daily routine of life of the child the parents are not going to bother about it. While this attitude in the majority of parents is a direct result of illiteracy, it is surprising how often it is displayed by the so-called educated parents. Time alone will bring about the change.

CONDITIONS IN PUBLIC HOSPITALS

In spite of the free treatment in our hospitals, people are most reluctant to go to a hospital. If the child has to be taken to a hospital it is looked upon as an *inevitable calamity*. The majority of parents will not think of taking their children to a hospital unless they are forced to do so. This, perhaps, is due to the hardships they experience in a public hospital. We cannot blame the hospital staff either, because our hospitals are overcrowded, and the hospital staff is worked to the limit of its capacity. For years our hospitals have been run entirely on a charitable basis, and the main object is to provide relief to the greatest number of patients with whatever meager resources we have at our command. Today we are occupied mainly with treating the disease rather than treating the person, and under existing circumstances there is very little scope to develop the patient-doctor understanding in our general hospitals. We are not in a position to give the child the understanding that a handicapped patient requires. Under these circumstances it is no wonder that many of them prefer to put up with a handicap in life rather than face the hardships in a hospital. Hospital authorities have their difficulties too. These will be considered subsequently.

FEAR OF SURGICAL TREATMENT

Where such is the attitude of the public toward hospitals, it is natural that the fear of a surgical operation should be dominant in the minds of most persons. The majority of persons will not think of submitting to a surgical operation unless it is a matter of life and death. Under these circumstances, a large number of operations have to be performed as emergency procedures to save life, and consequently the mortality rate is affected adversely. This risk is interpreted by the lay public as universal for all surgical operations, and, naturally, every surgical procedure is looked upon with extreme pessimism. No doubt, day by day, people are slowly getting over this fear, but it will take some time before it is dispelled completely and they learn to view the risks involved in their proper perspective.

ECONOMIC CONSIDERATIONS

The most vital consideration is the economic situation in the country. Henry D. Lloyd, an American social philosopher, wrote: "Nature is rich, but everywhere man, the heir of Nature, is poor." This is all the more true about India with our predominantly agrarian population and the vastness of our country. The Indian is probably the poorest man on this earth. The average per capita income in India is estimated at 67.5 rupees or £5 per annum, as against £76 in the United Kingdom and $398 in the U.S.A. This poverty is particularly revealed when we realize the uneven distribution of wealth in India. It is estimated that 35 per cent of the wealth in the country is owned by 5 per cent of the people; another 35 per cent is owned by 35 per cent of the population, and the remaining 30 per cent of the wealth is distributed among 60 per cent of the population. We often hear foreigners talking of the few needs of an Indian. It is said that an Indian can live on 7 rupees per month. But the fact is that he does not live on 7 rupees per month; he starves on 7 rupees per month. When the struggle for existence is so overwhelmingly difficult is it any wonder that the average Indian adopts an indolent and fatalistic attitude toward life? When the parents are struggling all their lives to keep body and soul together it is a mockery to talk to them about the physical, social or spiritual rehabilitation of one of the many children in the family. In

spite of free treatment in the hospital it may be difficult for the parents to attend the hospital. The father may have to lose a day's wage if he is to accompany the child to the hospital. The average Indian family has at least 3 children in the home, and the plight of an Indian housewife is very pitiable. She has to look after the house and the children, cook for the household and then rush to the hospital with the child, only to stand in a long queue for a couple of hours before the child is seen by the doctor. When she sees the doctor he may be able to persuade her to get the child's deformity corrected at the hospital, but when she is told that later on the child will require a brace or a shoe she is helpless and prefers to drop the idea of getting the child's handicap corrected. *The child will see to it when he grows* is her answer.

DISTANCE FROM HOME

India is not made up of cities like Bombay and Calcutta. It is made up of 700,000 villages where the majority of our population dwells. In most of these places there are no facilities for even routine minor medical aid. With very optimistic expectations, it would take perhaps another couple of generations before specialized treatment could be made available in these places. A sample survey conducted some time ago brought this factor into bold relief. Out of 167 cases of crippled children in different hospitals in the metropolitan cities in India, 141 came from the local respective cities; only 26 were from mofussil [provincial or rural] areas. Unless this fact is interpreted to signify that the number of crippled children in the mofussil area is small, one can imagine what must be happening to the rest of the crippled children who never come to the cities to get treatment.

IGNORANCE OF FACILITIES FOR TREATMENT

This probably is the most important single factor that is responsible for so much crippling in the country. Even in the Western World, the conception of rehabilitation of the *crippled is of comparatively recent* origin. It is no wonder then that in India we have not yet learned to appreciate its full importance. It is surprising how few, even in the medical profession, know to what extent a crippled child can be salvaged. Very often a child loses its only chance in life because the family doctor advises the *parents not to submit the child to new fads,* as he calls them, because in his opinion nothing can be done for the child. When this is the state of affairs in the medical world we can well imagine the ignorance among the lay public.

INSTITUTIONS

Having discussed the problem from the point of view of crippled children, it must also be examined vis-à-vis the institutions and the persons giving their services in the cause of crippled children, their number and the resources at their command. At present, the institutions in the country where a crippled child can avail himself of facilities for treatment are as follows:

1. General hospitals in the country (Government and Municipal)
2. Bai Jerbai Wadia Hospital for Children, Bombay
3. Children's Orthopaedic Hospital, Bombay
4. The Occupational Therapy School, Bombay (King Edward Memorial Hospital)
5. The Physiotherapy School, Bombay (King Edward Memorial Hospital)
6. The Hospital for Indian Medicine, Madras
7. A couple of clinics treating poliomyelitis
8. Private and semiprivate institutions

General Hospitals in the Country. In response to an inquiry regarding the facilities of treatment available to handicapped children in different states in India, the Directors of Public Health of 8 states supplied

the following information: In these states there are 26 general hospitals that have pediatric facilities. Of these hospitals, 13 have special wards for children; in others, the children are treated along with the adult patients in the same wards as adults. In these hospitals there are 22 surgeons who either have special qualifications in orthopaedics or are interested in orthopaedics.

If the conditions in a general hospital, in a city like Bombay, are to be taken as a basis, it will be seen that they scarcely meet the demands made on them. The number of beds on the orthopaedic side, the facilities available for treatment and the demands made on the staff are grossly out of proportion to the number of patients trying to seek treatment at these hospitals. The number of cases a visiting surgeon is expected to attend in a day can be in the vicinity of 100. Under these conditions it is hardly possible to do justice to each and every patient, and it cannot be said that we are able to give our patients the best we wish to give them. In hospitals having separate orthopaedic departments, the number of beds in an orthopaedic unit varies from 20 to 50. The demand on these beds is always heavy, most of them being occupied by emergency accident cases. Invariably the patients have to be discharged from the hospital not because they are ready to go out but because room has to be made for patients whose needs are more urgent.

Bai Jerbai Wadia Hospital for Children, Bombay. This hospital was founded in 1929 by the Wadia brothers in memory of their mother and is sponsored partly by the Bombay Municipality. It has a total of 135 beds, of which 36 beds are on the orthopaedic side. This was the first hospital in India to start an independent orthopaedic department under an orthopaedic surgeon In the hospital there is a well-equipped gymnasium for corrective exercises and rehabilitation work, and a swimming pool was added in 1946. A factory for manufacturing prosthetic appliances is maintained by the hospital (see prosthetic appliances). The small profits made by this factory are utilized, through the poor box fund of the hospital, to provide braces and appliances to poor children free of cost. Although this service is necessarily small, this is the only hospital that offers this service to the poor. All the treatment at this hospital is entirely free, regardless of the status and the income of the parents. All the members of the staff, except the residents, work in honorary capacities without remuneration of any kind whatsoever.

The Children's Orthopaedic Hospital, Bombay. This hospital is conducted by the Society for the Rehabilitation of Crippled Children, which was started by Mrs. Fatehma Ismail in 1947 with the help of some social workers and medical men. Originally started as a physiotherapy center for poliomyelitis patients, it has grown into a well-equipped orthopaedic hospital under the guidance of the late Dr. M. G. Kini. There are accommodations for 48 inpatients, and the hospital has a well-equipped physiotherapy department, with a swimming pool for underwater exercises Arrangements for the education of children during the time they are attending the hospital also are made by the hospital. As an institution specializing in a particular type of work and utilizing its entire resources in a particular direction, it should be in a position to provide more adequate facilities than a general hospital. The hospital has a full-time paid staff. Charges for treatment are adjusted according to the paying capacity of the parents.

The Occupational Therapy School, Bombay. This school was started at the King Edward VII Memorial Hospital, Bombay, in 1950, by Mrs. Kamala Nimbkar, herself a trained occupational therapist, who worked in the hospital for a year to convince the hospital staff and the authorities of the place and the advantages of occupational therapy in a hospital. She has undertaken to work as Honorary Director of the school for a period of 5 years, in addition to providing

equipment for the school. Mr. Nimbkar has donated 30,000 rupees toward the building cost of the school.

This is the only occupational therapy school in the East, and countries outside India have started to send their students for training at this school. It has facilities for the training of 30 students, 15 in each of the 2 classes. In addition to providing occupational therapy to the patients of King Edward Memorial Hospital, it extends its facilities to other institutions in the city. The first batch of students will be graduating this month [November, 1952].

The Physiotherapy School, Bombay. The Physiotherapy Department at King Edward Memorial Hospital, Bombay, was started in 1949 under the guidance of a poliomyelitis team of the World Health Organization which visited India that year. Now [1952] an attempt is being made to develop this into a physiotherapy school. The school will be sponsored jointly by the Government of India, the Government of Bombay and the Bombay Municipality. The World Health Organization has undertaken to provide for the first 2 years the services of 2 qualified physiotherapists, one from the U.S.A. and the other from the United Kingdom. One of them has arrived, and the other is on the way. A substantial part of equipment for the department also will be provided by the W.H.O. It is proposed to begin the admission of students for the physiotherapy course from June, 1953.

The Hospital of Indian Medicine, Madras. This institution is unique in the sense that it has a well-developed system of treatment based exclusively on the Indian system of medicine for the care of cripples. Massage with herbal extracts forms the major part of the treatment program In other treatments also the emphasis is laid on the indigenous herbal medicines.

Poliomyelitis Clinic, Hyderabad. This clinic is run on the same lines as the Society for the Rehabilitation of Crippled Children in Bombay As the name indicates, this clinic concentrates mainly on poliomyelitis cases in children.

Private and Semiprivate Institutions. In addition to the above institutions, there are a number of other institutions in the country that care for crippled children. As most of them depend for their maintenance either on the public or private charities, their scope of work is necessarily limited. Some of them receive grants in aid from the local governments. Most of these institutions depend on the services of honorary physicians who pay periodic visits to the institutions.

PROSTHETIC APPLIANCES

The manufacture and supply of prosthetic appliances to the handicapped has not made much progress in India as yet. There are one or two firms in most of the larger cities that undertake to supply prosthetic appliances. Since this type of work does not hold out a decent margin of profit, there is not much enthusiasm on the part of commercial firms to develop this line.

In Bombay, four commercial firms and the orthopaedic factory maintained by the Jerbai Wadia Hospital for Children manufacture prosthetic appliances. Recently one of the firms imported a British technician for this purpose, and the quality of the appliances turned out compares favorably with that of British or American manufacture. The factory at the Jerbai Wadia Hospital is run by the hospital and has a full-time staff. Since the hospital runs the factory there is no commercial outlook. In addition to serving the hospital, they also accept orders from other hospitals and private medical practitioners They executed over 700 orders last year. The quality of the work is fairly satisfactory, but the finish is not quite perfect. This is due to the fact that the main object is to make the appliances as cheaply as possible so as to bring them within easy reach of the poor patients, who form over 90 per cent of the children attending the hospital. The service for the adjustment, the repair and the maintenance of the appliances is

given ungrudgingly and free in most of the cases, unless it involves considerable expense to the hospital.

The artificial limbs are manufactured and fitted at the Army Artificial Limb Centre near Poona. With the permission of the Army Headquarters, they accept orders from civilians also. The staff, it is understood, was trained by Hangers, of Roehampton, England, and for the first few years the factory was under their supervision. Once they accept a patient, they see to the manufacture, the fitting of the limb and the complete rehabilitation of the amputee until he is able to use the limb properly and get about. Facilities for the admission of the amputee to the hospital during the period of training are available on payment.

Although almost all types of prosthetic appliances are available in India today, their cost is still high and beyond the reach of most of the people. Similarly, services for the adjustment, the fitting, the repair and the maintenance of the appliances, once they are sold, are not very satisfactory. It will not be possible for the majority of patients to avail themselves of prosthetic appliances until some solution can be evolved to get over these two difficulties.

ORTHOPAEDIC SURGERY IN INDIA

The status of orthopaedic surgery as a speciality is not yet sufficiently appreciated in most parts of the country. In most of the hospitals the general surgeons are doing routine orthopaedic work and, as everywhere else in the world, they are unwilling to part with orthopaedic work. Day by day this opposition is dying out, and now, at least in the cities like Bombay, Calcutta, Madras and Delhi, attempts are being made to establish orthopaedic departments in general hospitals under an orthopaedic surgeon who restricts his practice to his speciality only, both outside and inside the hospital. Mr. R J. Katrak, Honorary Orthopaedic Surgeon at the King Edward Memorial Hospital in Bombay, who restricts his practice to orthopaedics only, was the pioneer in this field, having established the first orthopaedic department in India at the Jerbai Wadia Hospital for Children in 1936. In India there are now about 15 orthopaedic surgeons who were specially trained abroad in this field and confine their work to orthopaedics alone; there are probably as many others who are doing orthopaedic work as a side interest in addition to general surgery.

ANSWER TO THE PROBLEM

The solution of a problem with such gigantic difficulties cannot be easy. However, a beginning has to be made somewhere. The first thing is the enumeration of handicapped children in the country. The easiest way to do this is to instruct every school teacher to report a handicapped child to the nearest Government medical officer, who, in turn, should examine the child and send his report to the Surgeon General's office. This way we will be able to get an idea of the number of handicapped among the school children, at least, and in about 5 years the records will be fairly comprehensive. Since the people will not come to the big hospitals in the cities for treatment, facilities must be carried to them. The only way to do this is to provide a large number of small institutions widely scattered in the country so as to be within easy reach of the people. They should not be run as a small part of a big hospital but should function as independent units in close liaison with them. All that is required in each unit is the provision of a few beds, a small operation theater, a small x-ray unit and a physical medicine and rehabilitation setup Most of the work can be managed by the local medical practitioners under the guidance and direction of an orthopaedic surgeon from a near-by hospital who visits the place periodically. With this setup it should be possible to handle about 80 per cent of the handicapped children. Our main object should be to provide that type of care which is not possible in an aver-

age home and for which it is not necessary to go to a big hospital. Children requiring special treatment will be sent to the hospital. But the majority of crippled children are not ill patients, and for them there is no need for elaborate hospitals with many trained employees per head. This, in turn, will reduce the daily cost per patient. Moreover, in these days when our hospitals are overcrowded, these small clinics will relieve them of the care of children no longer acutely ill but needing only supervision by trained personnel.

In this way, the treatment facilities at present available only in metropolitan cities will be decentralized, and treatment can be carried to every home in India, even in the farthest corner of the country.

The rehabilitation of the handicapped child, his psychological needs, the implications in regard to education, child upbringing and vocational guidance are other major problems that have received scant attention in this country so far. But their need is beginning to be appreciated, and amenities in this field are trickling through slowly.

This far we have come: the sun has risen, but it is still on the horizon.

28

Use of the Extra-Short Smith-Petersen Nail for Subtrochanteric Osteotomy in Children

W. Compere Basom, M.D., M.S. (Or.)*

The Smith-Petersen nail plate has proved its value in the management and the internal fixation of intertrochanteric and subtrochanteric fractures and also in osteotomies in adults. The standard Smith-Petersen nail can easily be shortened to lengths of from 1 in., 1¼ in. on up to 3 in., which is the standard shortest length. The advantage of the separate nail and plate is that the nail can be put into proper position through the trochanter into the femoral neck first, then the osteotomy can be done, and by attaching the plate thereafter the surgeon has full control of the short upper fragment.

The Smith-Petersen nail has serrated edges about its head; the trochanteric anchor plate is merely the shortest standard size, and it has a serrated well in its upper end which fits to the nail preventing rotation. The bolt is used merely to anchor the plate to the nail first, then the correction can be obtained following the osteotomy. Then the plate is attached to the femoral shaft, and fixation of the corrected postosteotomy position is then maintained.

The anchor plate can be bent to any angle desired prior to this so that the maximum amount of correction can be secured.

A simple transverse osteotomy can be used in children. The osteotomy site is filled with bone chips.

This material was presented as a gadget exhibit at the 23rd Annual Meeting of The

* El Paso, Tex.



Fig. 1. Roentgenogram of the pelvis showing a coxa vara position



Fig. 2. Roentgenogram showing initial osteotomy, merely a simple transverse type with angulation of the lateral aspect and fixation by the short nail and the plate arrangement.



FIG. 3. Roentgenogram showing the fully united osteotomy site for the correction of the coxa vara. It is planned to remove the nail and the plate in the near future.

American Academy of Orthopaedic Surgeons, *January 28 to February 2, 1956, at* Chicago, Ill. The purpose of the author here is merely to record the essentials of the exhibit.

The exhibit emphasized the fact that the extra-short Smith-Petersen nail can be used as an internal fixation device to maintain the correction of subtrochanteric osteotomy in children. It could be used for fractures, should that be indicated.

BIBLIOGRAPHY

Albee, F. H., and Preston, R. L.: Injuries and Diseases of the Hip, New York, Hoeber, 1937.

Anderson, R., McKirtlin, W. B., and Burges, E.: Intertrochanteric fractures: non-operative, castless and ambulatory method of treatment,

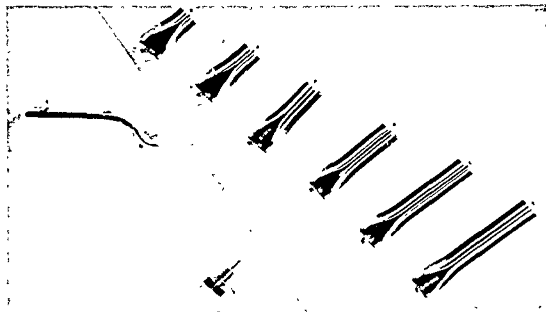


FIG 4 Photograph of the *extra-short Smith-Petersen* nails The top nail is 2¾ inches in length. The bolt fits the standard-size screws which the driver utilizes in the Smith-Petersen nail It has a small guide on the end that facilitates its attachment to the nail, *and the guide* has a suitable d[illegible] fit inside the cannula. The anchor plate is merely the shortest standa[illegible] angle can easily be bent by the use of a spec[illegible] or by the use [illegible] a hammer.

Babb, F. S., Ghormley, R. K., and Chatterton, C. C.: Congenital coxa vara, J. Bone & Joint Surg. 31A:115, 1949.

Bartels, R. N.: The treatment of intertrochanteric fractures by blade plate internal fixation, Bull. Linn County M. Soc. 13:11, 1945.

Basom, W. C.: The use of the extra-long trochanteric plate for severely comminuted fractures of the upper third of the femoral shaft and the subtrochanteric area, J. Internat. Coll. Surgeons 25:348-355, 1956.

Blount, W. P.: Blade plate internal fixation for high femoral osteotomies, J. Bone & Joint Surg. 25:319, 1943.

Breck, L. W.: Corrective osteotomies held in position by properly bent Vitallium plates, Quart. Bull. Northwestern Univ. M. School 22:128-131, 1947.

Buckwalter, J. A.: The immediate treatment of trochanteric fractures, Am. Pract. & Digest. Treat. 5:960-962, 1954.

Cleveland, M., Bosworth, D. M., and Della Pietra, A.: Subtrochanteric osteotomy and splint fixation for certain disabilities of the hip joint, J. Bone & Joint Surg. 33A:351, 1951.

Cleveland, M., Bosworth, D. M., and Thompson, F. R.: Intertrochanteric fractures of the femur; survey of treatment in traction and by internal fixation, J. Bone & Joint Surg. 29: 1049-1067, 1947.

Compere, E. L., Wallace, G., and Lee, J.: Materials for internal fixation of intra-capsular fracture of the neck of the femur, Arch. Surg. 44:327-338, 1942.

Cozen, L., Brockway, A., and McMaster, P. E.: Operative Orthopedic Clinics, Philadelphia, Lippincott, 1955.

Evans, E. M.: Trochanteric fractures; a review of 110 cases treated by nail-plate fixation, J. Bone & Joint Surg. 33B:192, 1951.

Farill, J.: Nuestra conducta en las fracturas intracapsulares del cuello del femur, Gac. méd. México 73:85, 1943.

Golding, F. C.: Congenital coxa vara, J. Bone & Joint Surg. 30B:160, 1948.

Haas, S. L.: Lengthening of the femur with simultaneous correction of coxa vara, J. Bone & Joint Surg. 15:219, 1933.

Haboush, E. J.: A universal nail: instruments for the treatment of fractures of the femur and biomechanical considerations, Bull Hosp. Joint Dis. 15:223-242, 1954.

Herz, J. R., Breck, L. W., and Basom, W. C.: A lock screw for attaching the intertrochanteric plate to the Smith-Petersen nail, J. Bone & Joint Surg. 30A:1008-1009, 1948.

Horowitz, T.: The postero-lateral approach in the surgical management of basilar neck, intertrochanteric and subtrochanteric fractures of the femur, Surg., Gynec. & Obst. 95:45-50, 1952.

Jewett, E. L.: One piece anglenail for trochanteric fractures, J. Bone & Joint Surg. 23:803-810, 1941.

Key, J. A., and Conwell, H. E.: Fractures, Dislocations and Sprains, St. Louis, Mosby, 1951.

Kleinberg, S.: Fractures of the neck of the femur; surgical technique for reduction and internal splinting by direct visualization, New York J. Med. 44:2460-2467, 1944.

LeMesurier, A. B.: Developmental coxa vara, J. Bone & Joint Surg. 30B:595, 1948.

McLaughlin, H. L., and Garcia, A.: An adjustable fixation device for the hip, Am. J. Surg. 89:867-871, 1955.

Magnuson, P. B., and Stack, J. K.: Fractures, Philadelphia, Lippincott, 1949.

Moore, A. T.: Blade plate internal fixation for intertrochanteric fractures, J. Bone & Joint Surg. 26:52, 1944.

Moore, M., Jr.: Treatment of trochanteric femoral fractures with special reference to complications, Am. J. Surg. 84:449-452, 1952.

Murray, R. C., and Freu, J. F. M.: Trochanteric fractures of the femur, J. Bone & Joint Surg. 31B:204, 1949.

Overton, L. (quoted by Stuck): The Internal Fixation of Fractures, Springfield, Ill., Thomas, 1947.

Smith-Petersen, M. N., Cave, E. F., and Vangorder, G. W.: Intracapsular fractures of the neck of the femur, Arch. Surg. 23:715-759, 1931.

Speed, J. S., and Knight, R. A.: Campbell's Operative Orthopaedics, ed. 3, St. Louis, Mosby, 1956.

Speed, J. S., and Smith, H.: Campbell's Operative Orthopaedics, St. Louis, Mosby, 1949.

Taylor, G. M., Neufeld, A. J., and Jaryan, I.: Internal fixation for intertrochanteric fractures, J. Bone & Joint Surg. 26:707, 1944.

Thornton, L., and Sandison, C.: The treatment of trochanteric fractures of the femur; two new methods, Piedmont Hosp. Bull. 10:21, 1937.

Venable, C. S., and Stuck, W. G.: The Internal Fixation of Fractures, Springfield, Ill., Thomas, 1947.

Watson-Jones, R.: Fractures and Joint Injuries, Baltimore, Williams & Wilkins, 1955.

29

Fixation of Beak Fractures of the Os Calcis by Stapling

H. L. GREENE, M.D.*

The use of staples in orthopaedic surgery has been popularized by Dr. Robert E. Burns† and Dr. Walter P. Blount. Recently a Vitallium staple was produced that gives far greater immobilization and rigidity by a bridge at each angle of the staple at its base. This staple has been described by Dr. Blount.‡ The following is a report of a severe type of beak fracture of the os calcis which was treated successfully by fixation with the Vitallium staple:

The history of this man revealed that on July 2, 1955, he fell a distance of 14 feet and landed on his left foot on the concrete. He entered a local hospital where roentgenograms were taken; then he was transferred to the Madison General Hospital for treatment on July 5. On July 7 an open reduction was performed, the fragments manipulated and approximated by the use of an Albee tenaculum. After satisfactory reduction had been accomplished, a staple was driven into the two major fragments. He was discharged from the hospital on July 10, wearing a short-leg cast and walking on crutches. The plaster cast was removed on August 17, and he was allowed some weight-bearing on the foot 3 weeks later. He returned to work on September 28, and since that time has been working 8 hours a day on his feet and sometimes puts in overtime of an hour or more. He works for the Water Department in his home town. His work requires him to jump off and on a dump truck and to dig ditches, as well as all other types of labor involved in the City Water Department. He

* Madison, Wis.

† Personal communication

‡ Association of Bone and Joint Surgeons Meeting, Minneapolis, Minnesota, April, 1956.



FIG. 1. Roentgenograms of left foot taken on admission to hospital.

does this without any complaint. He states that he can dance without any difficulty. He has no complaint of swelling about the foot. He wears ordinary shoes. He notices some discomfort in the region of the os calcis only during changes of weather. When he was last seen on February 9, 1956, he had normal ankle motions, with the exception of a 5° limitation of supination.



FIG. 2. Roentgenograms showing final result.

30

Wire Self-Retaining Retractor

WILLIAM MINOR DEYERLE, M.D.*

With the idea in mind of obtaining maximum exposure with minimal retractor material in the depth of the wound while removing plantar neuromas, I began using No. 24 and No. 26 stainless steel sutures, as shown in Figure 3. These can be placed, using the largest size curved cutting needle, in such a way that when traction is placed upon the wire loops it does not cut the skin.

A sponge can be placed under the edge as it comes past the wound, and the portion in the depth of the wound is placed so that it will not pull against the skin to any material extent. Then the wire can be looped and retracted with only the use of a sponge. This has been refined to some extent by making grooved and serrated protectors for the wound edge, as shown in Figure 1. It can be refined further by the use of the 4¾-

* Richmond, Va.

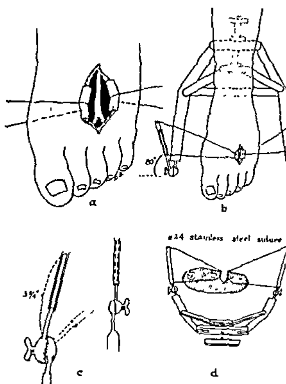


FIGURE 2

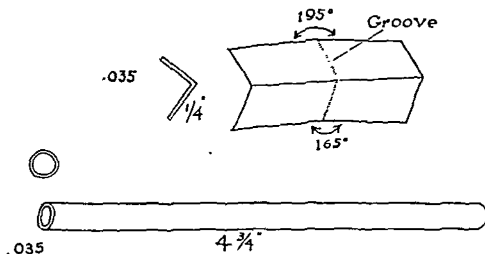


FIGURE 1

inch metal sleeves to prevent the wire cutting into the gloved hand and may be held in place by the modified Kirschner wire retractor, as shown in Figures 1, 2 and 3. This allows excellent exposure and visibility which can either be held manually or self-retained using the Kirschner wire retractor.

In removing the sesamoid bone in a McBride bunionectomy the same procedure may be used by placing the suture in such a way that one retracting suture is against the metatarsal and the other is threaded through the attachment of the sesamoid bone so that when it is retracted the sesamoid bone is pulled into the space between the 1st and the 2nd metatarsals. The same wire retractor may be used very satisfactorily either to spread or coapt the spinous processes in spinal surgery. In this instance the wire is looped around the spinous process or placed under the lamina. The refinements mentioned above can be obtained from the Zimmer Manufacturing Company in Warsaw, Indiana.

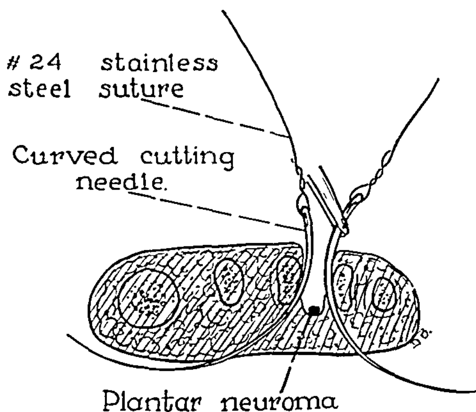


FIGURE 3

Index